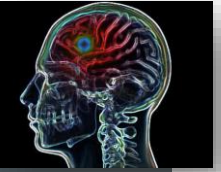


NEUROPATHOLOGY

INTRACRANIAL HEMORRHAGE, EDEMA, HERNIATION

HAJNALKA RAJNAI





INTRACRANIAL HEMORRHAGE

ETIOLOGY:

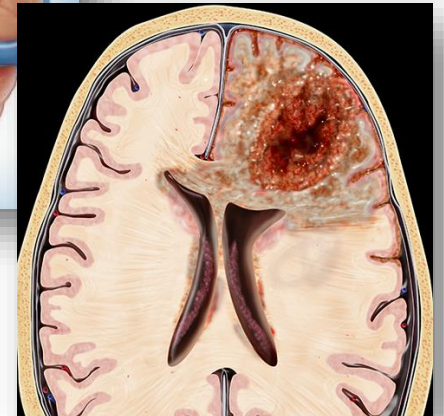
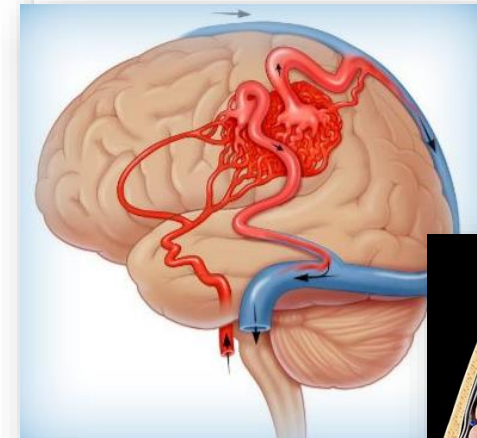
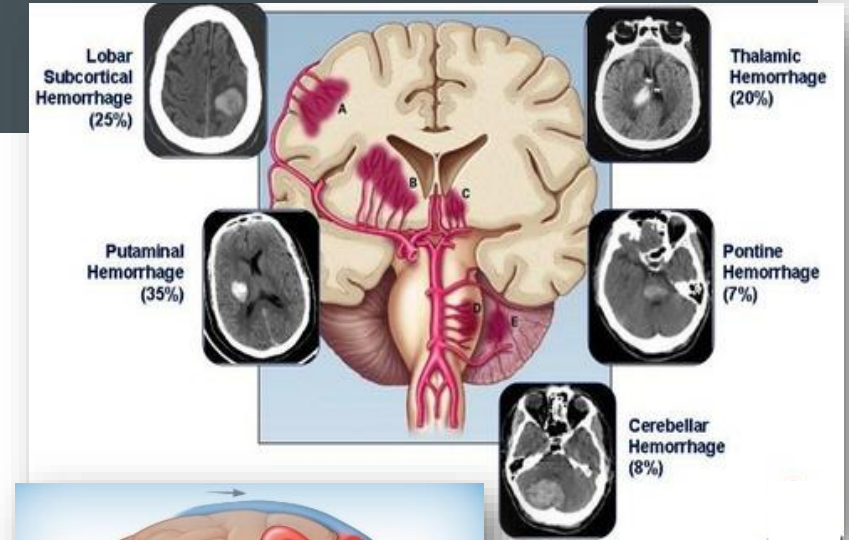
I. Vascular wall injury

- Hypertension
- Protein deposition
- Trauma

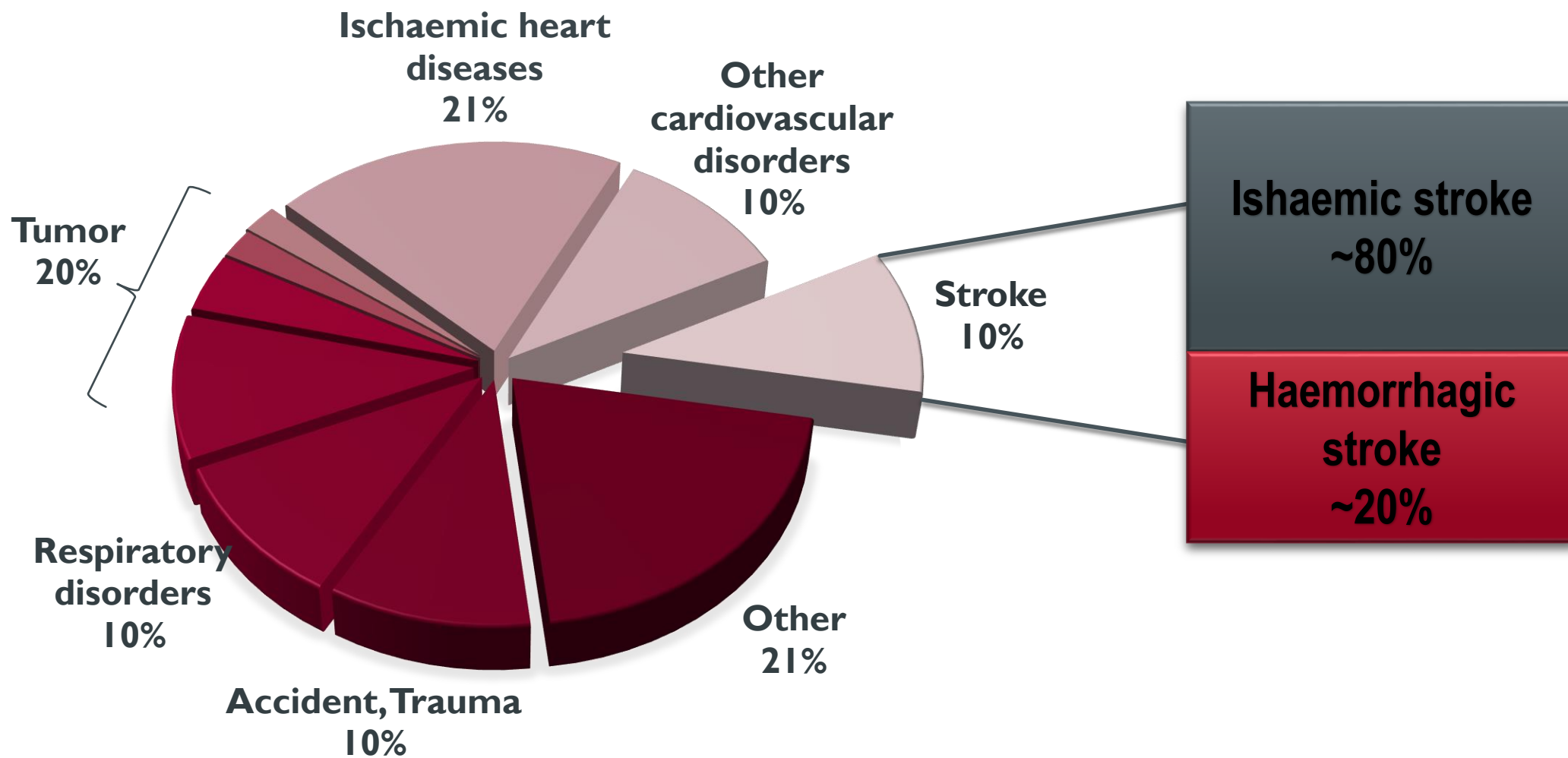
II. Primary structural lesions of the vessels

III. Tumors

IV. Hematological disorders



MORTALITY, EUROPEAN CARDIOVASCULAR DISEASE STATISTICS 2013



LOCATION:

I. Intraparenchymal hemorrhage

- Hypertension
- Tumor

II. Subarachnoideal hemorrhage

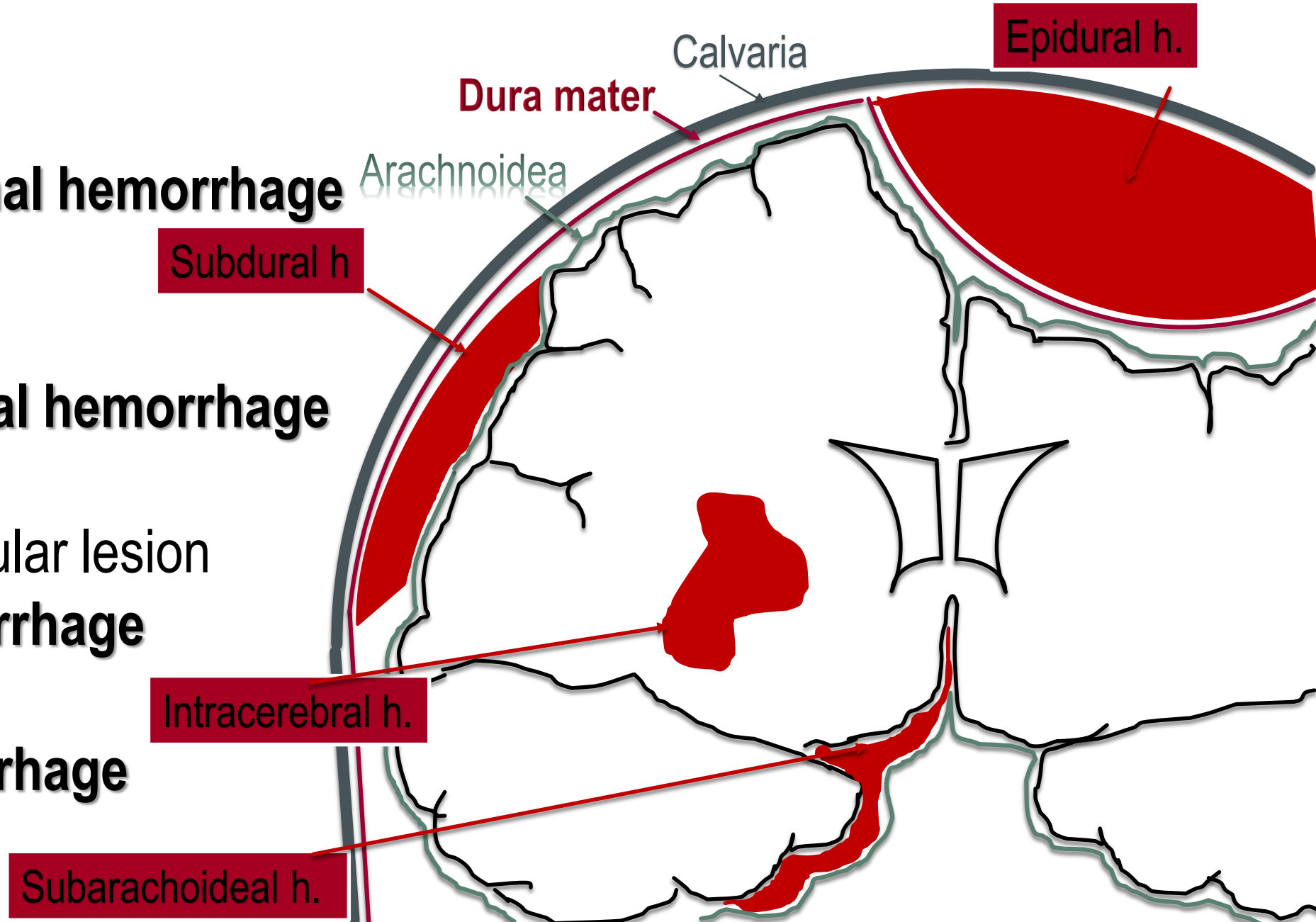
- Aneurysma
- Primary vascular lesion

III. Subdural hemorrhage

- Trauma

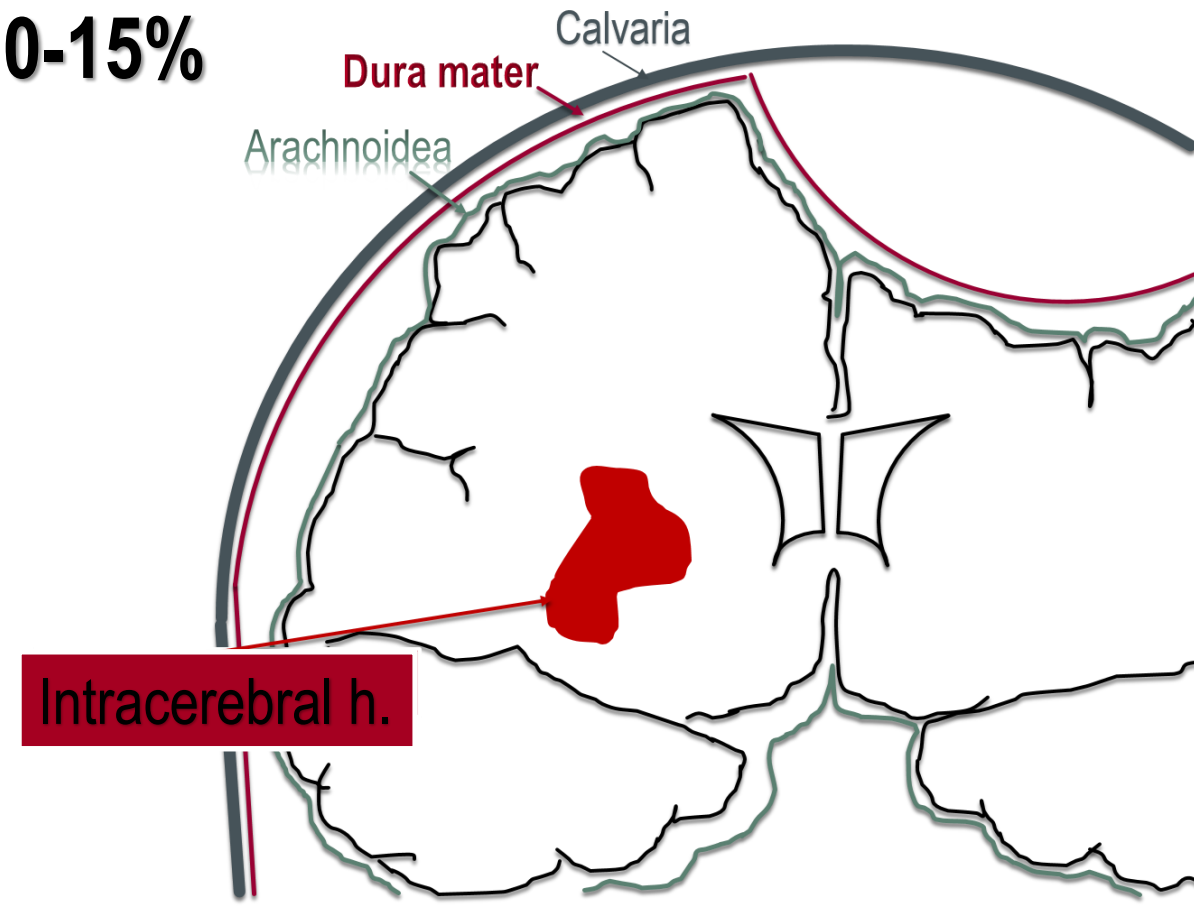
IV. Epidural hemorrhage

- Trauma



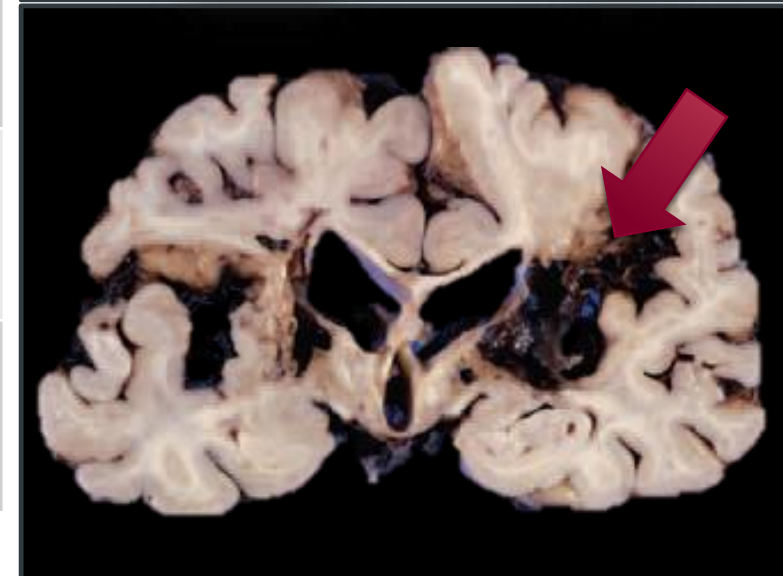
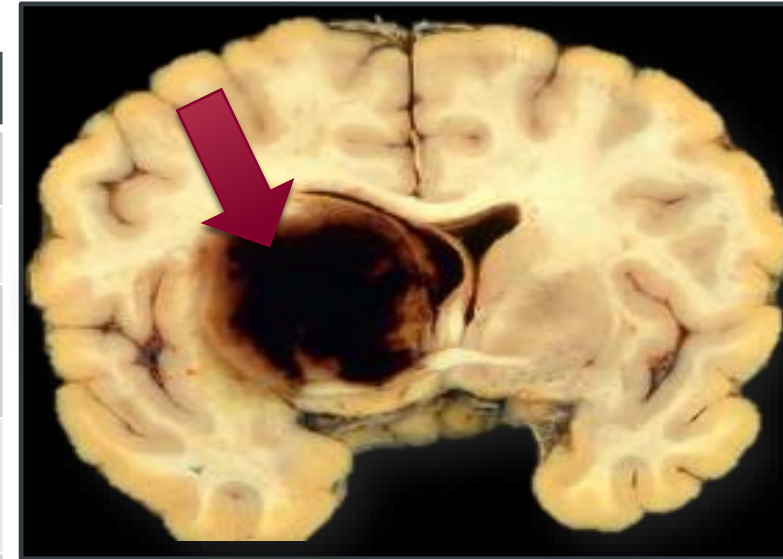
INTRAPARENCHYMAL BRAIN HEMORRHAGE

1. Hypertension ~ 50%
2. Cerebral amyloid angiopathy ~10-15%
3. Tumor ~8-10%
4. Vascular malformations ~5%
5. Trauma
6. Hematological disorders
7. Old age



Morphology

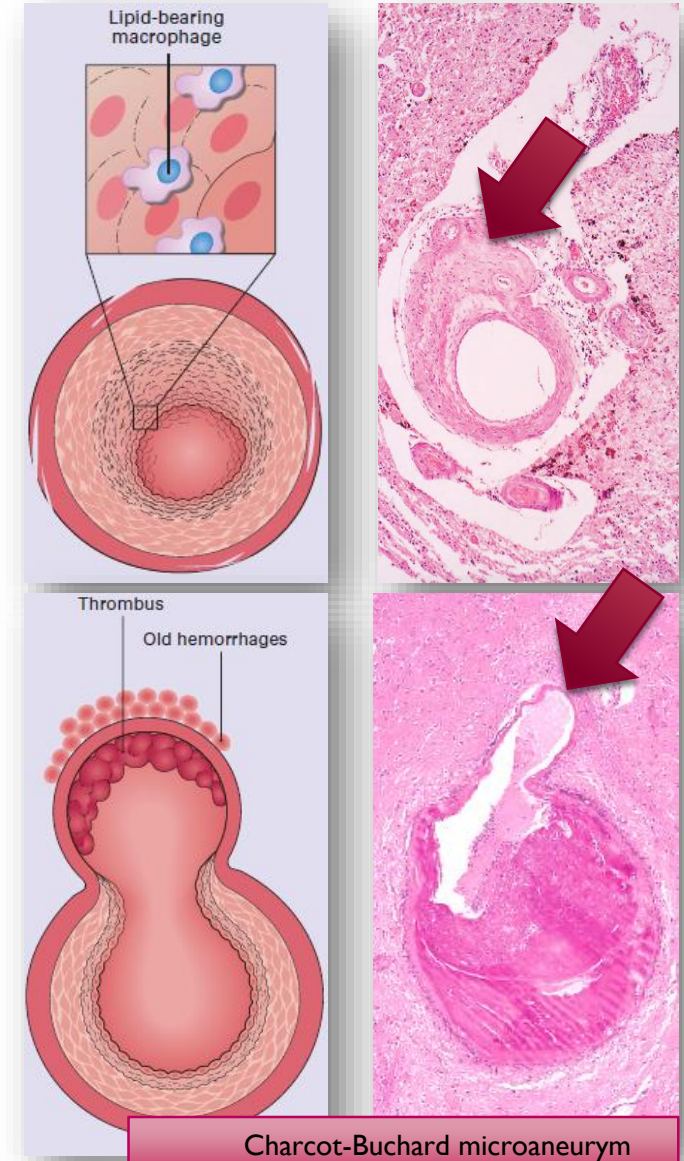
Time	Macroscopy	Microscopy
Seconds		Rupture
Minutes	Bright red hematoma	Extravasation
Hours	Perifocal edema, compression	Erythrocyte lysis edema, ischaemia
2-3 days - week	Brown hematoma	Hemosiderin-laden macrophages Astrocytosis
Weeks-Months	Friable brown hematoma	Organisation Phagocytosis of necrotic tissue
Months-1 year	Cyst containing brownish liquid	Cyst lined with glial cells Residual hemosiderin Macrophages
Yers	Cyst with brownish wall containing clear liquid	Cyst lined with glial cells Residual hemosiderin Macrophages



Hypertensive brain hemorrhage

Intraparenchymal arteriolar walls are weakened

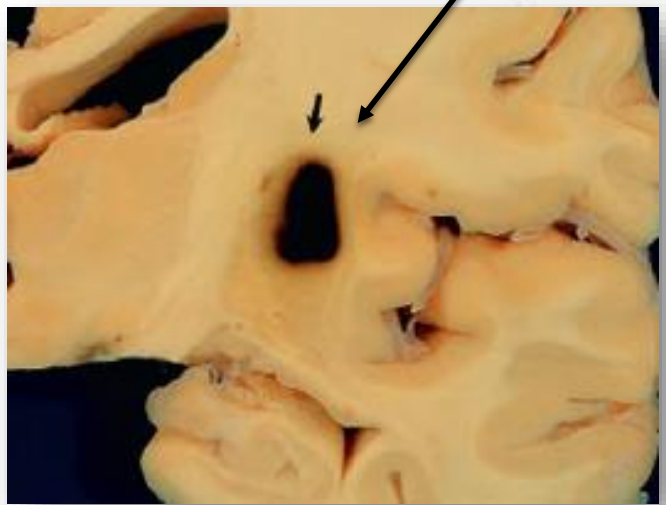
- Hyaline arteriolar sclerosis
- Fragmentation of elastica membrane
- Focal dilation of small vessels
 - Charcot-Buchard microaneurysms



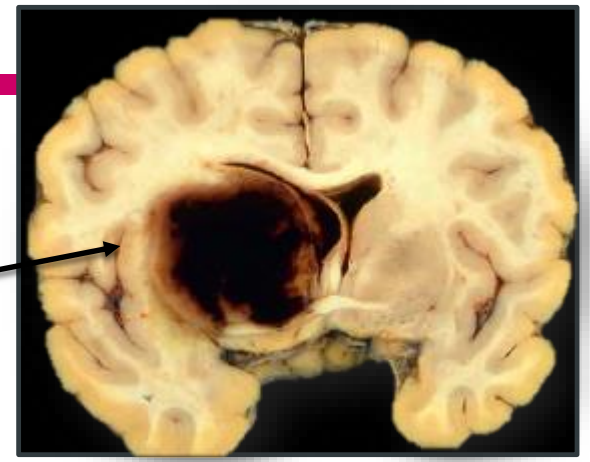
**Lobaris
Subcorticalis
25%**



**Putamen
35%**



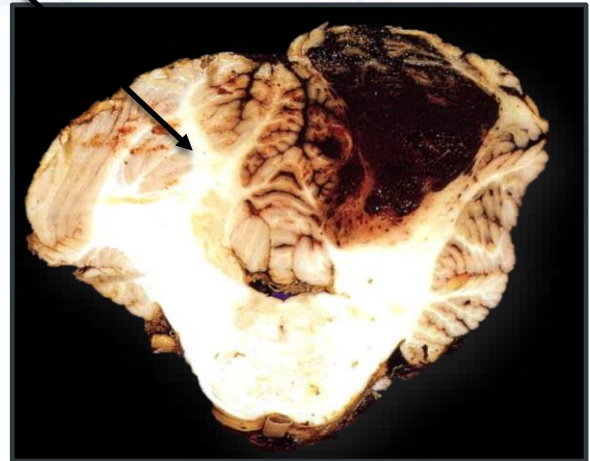
**Thalamus
20%**



**Pons
7%**



**Cerebellaris
8%**



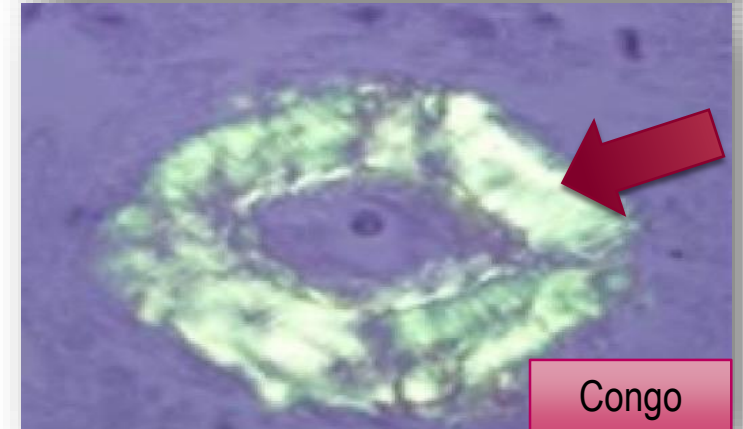
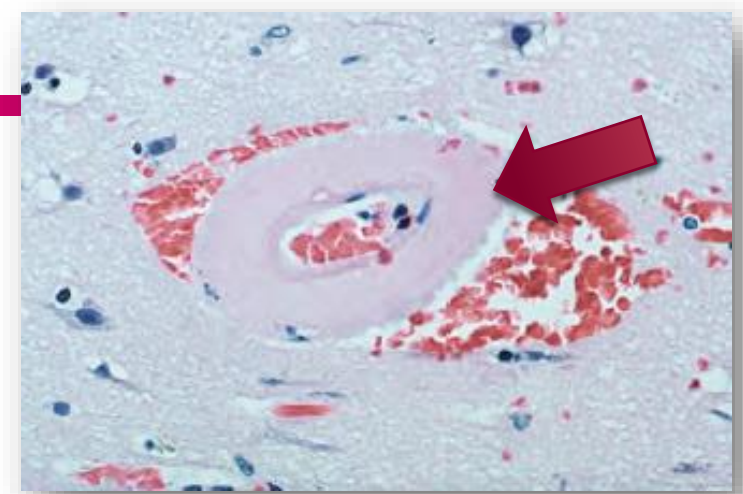
Cerebral amyloid angiopathy (CAA)

1. Sporadic/age-associated CAA

- Linked to Alzheimer's disease
- A β amyloid deposition
 - Disappearance of smooth muscle

2. Familial CAA

- Younger age
- Autosomal dominant
- Amyloid precursor protein mutation



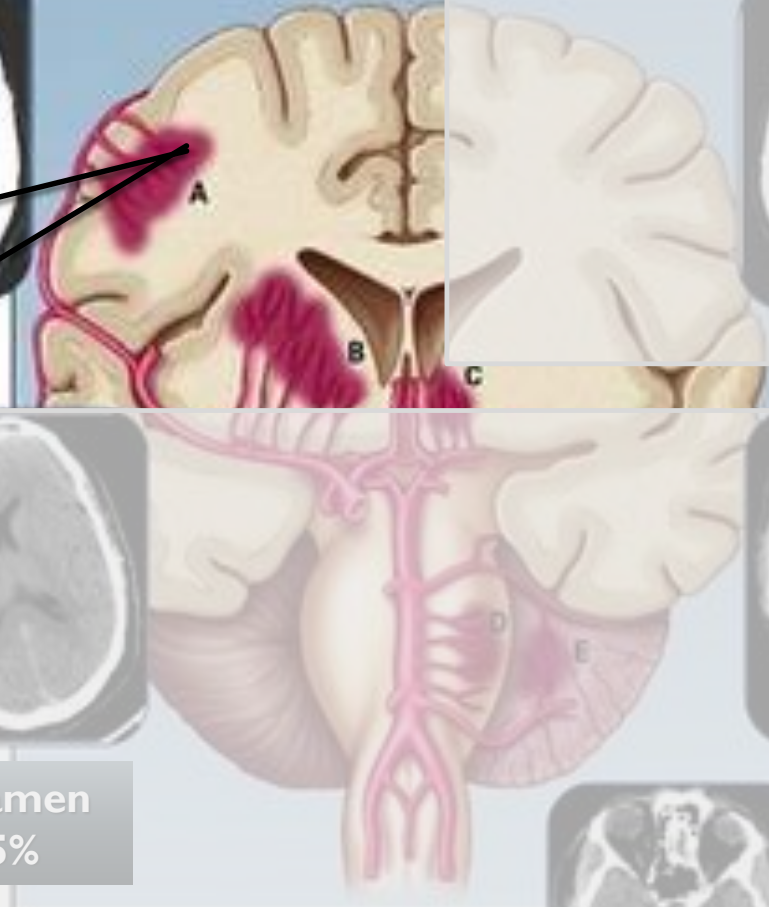
Congo



Anti- A β



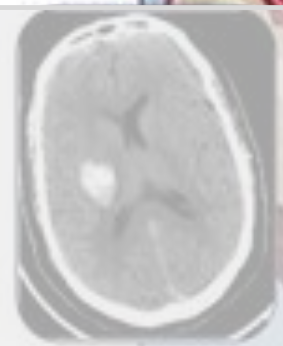
**Lobar
Subcortical
25%**



**Thalamus
20%**



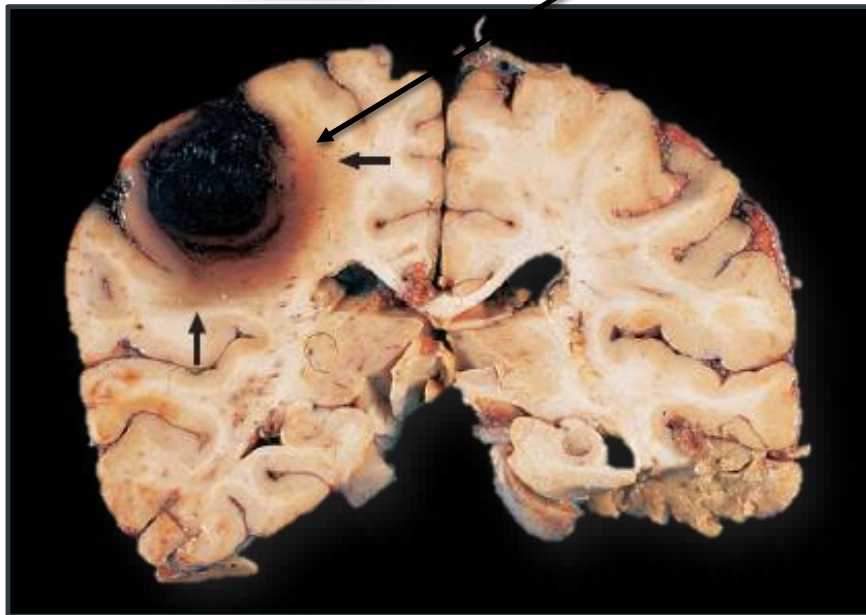
**Pons
7%**



**Putamen
35%**



**Cerebellaris
8%**



Neoplastic lesions

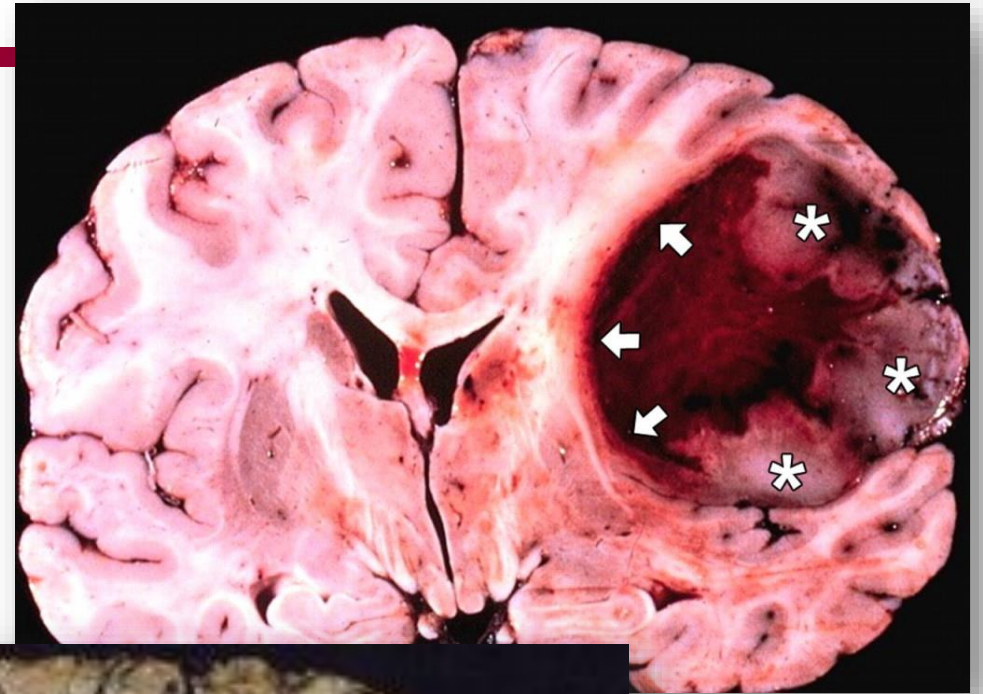
- No specific localisation
- Atypical, abnormal vessels

1. Primary CNS Tumors

- Glioblastoma
- Meningeoma
- Pituitary adenoma

2. CNS metastasis

- Melanoma malignum
- Renal cell cancer



SUBARACHNOIDEAL HEMORRHAGE

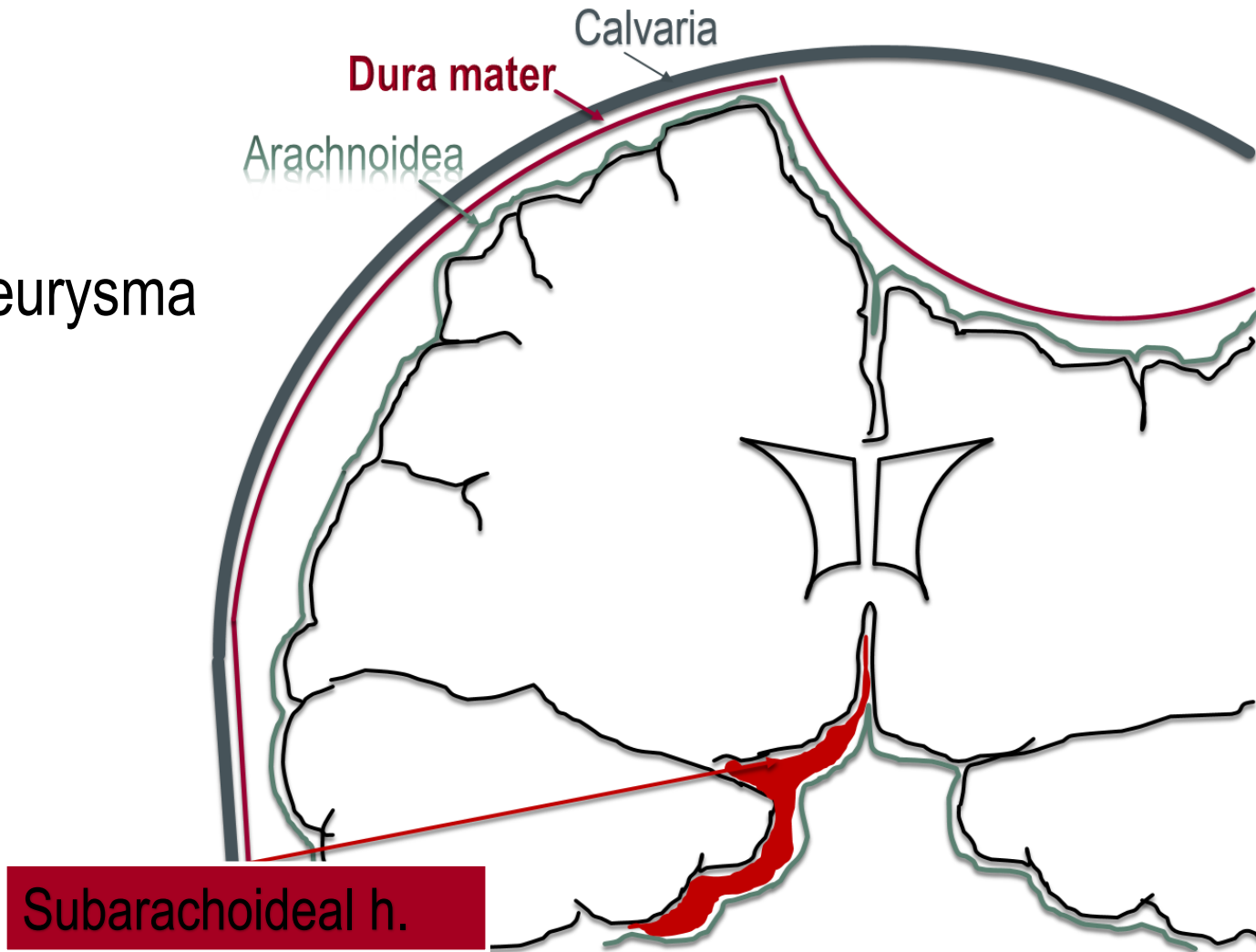
1. Aneurysm

- Saccular /berry/ aneurysma
- Fusiform /atherosclerosis/ aneurysma
- Infektív aneurysma

2. Vascular malformations

3. Trauma

4. Infections

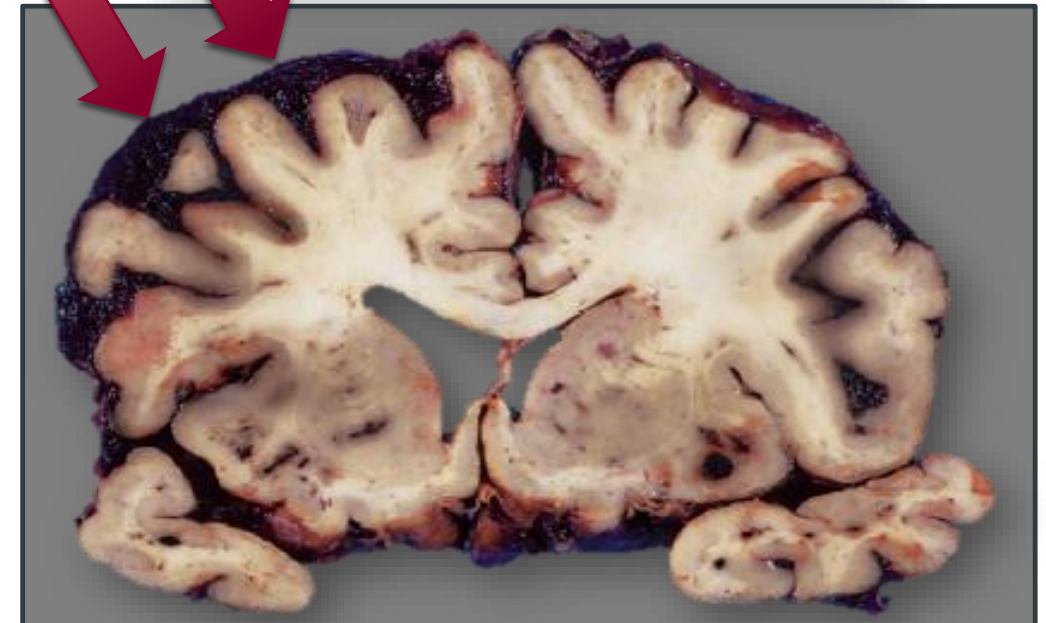
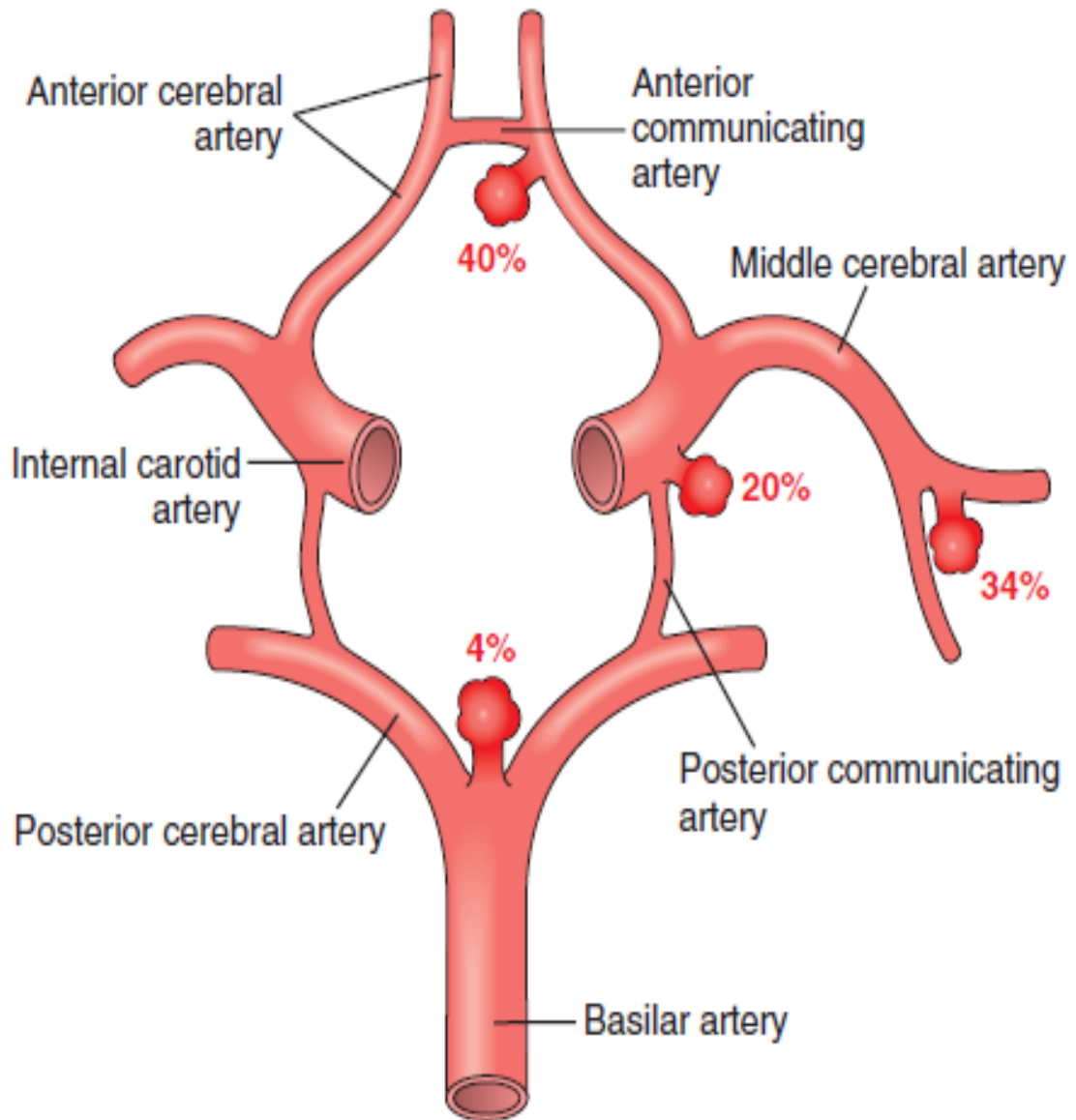


Subarachnoid Hemorrhage and Aneurysm

Saccular /Berry/ aneurysm

- Congenital – defects in the vessel media
- **Risk factors**
 - Hypertension
 - Systemic vasculopathy
 - Connective tissue diseases
 - Polycystic kidney disease
- Probability of rupture increasing with size





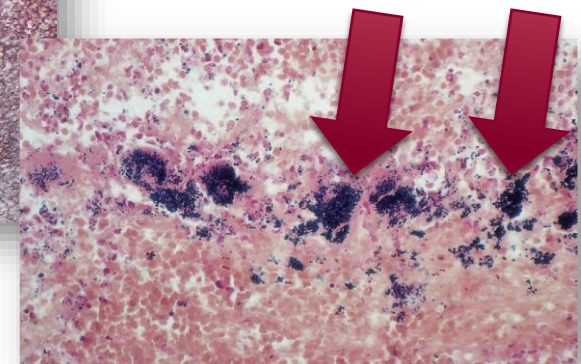
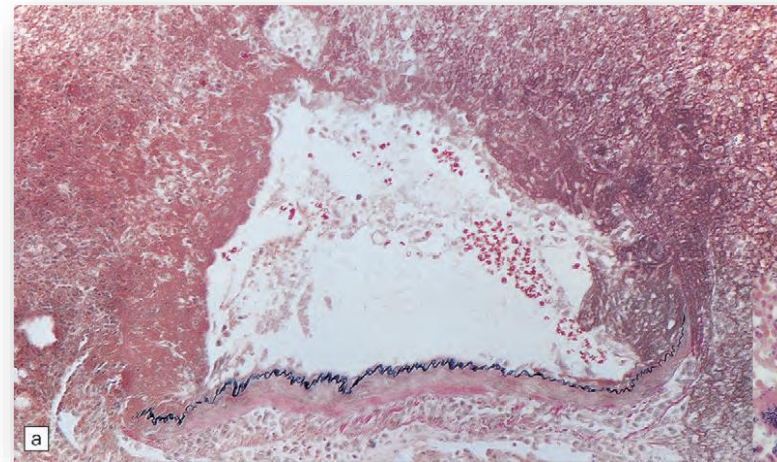
Fusiform aneurysm

- Atherosclerotic origin
 - Most commonly involve basilar artery
- Complications
 - Cranial nerve compression
 - Occlusion
 - Rupture - Rare



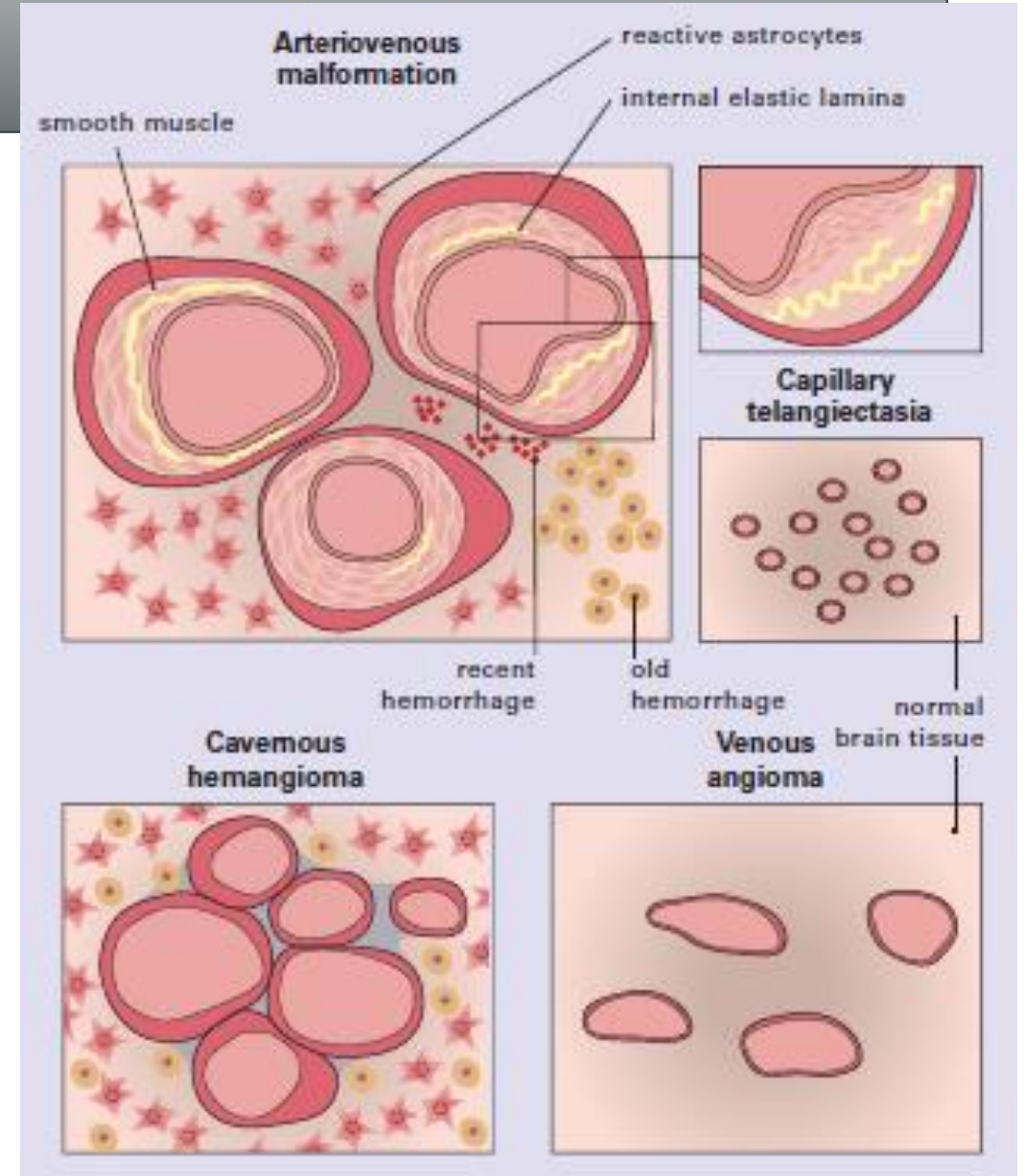
Infective /mycotic/ aneurysm

- Inflammation weakens the vessel wall
- Bacterial, fungal infection
 - Hematogenous origin (Endocarditis)
 - Local infection (Meningitis)
- Distal branches of cerebral vessels



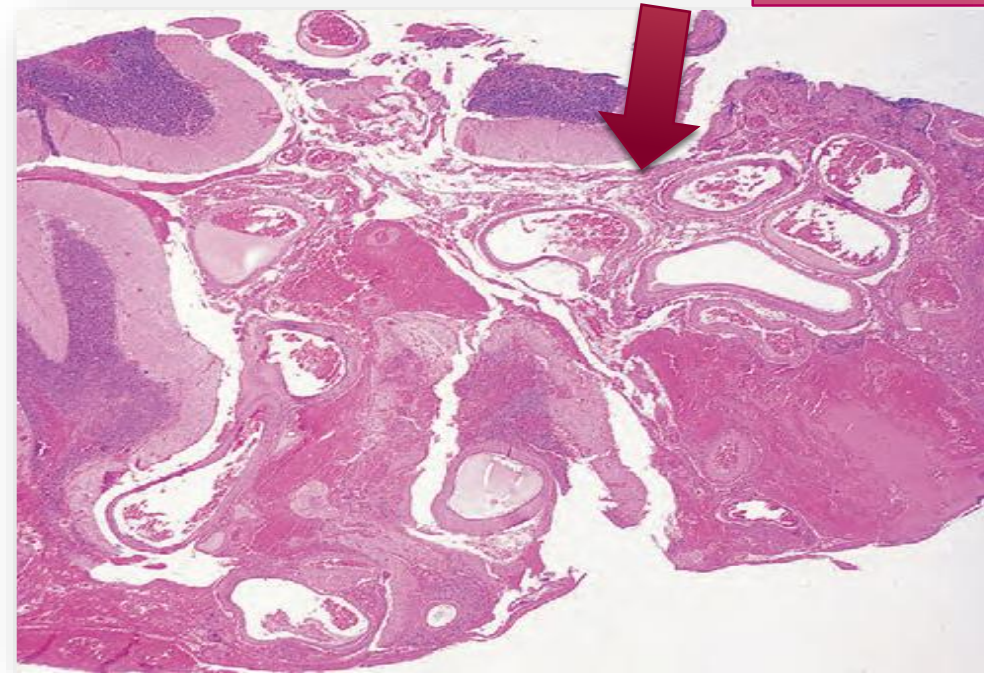
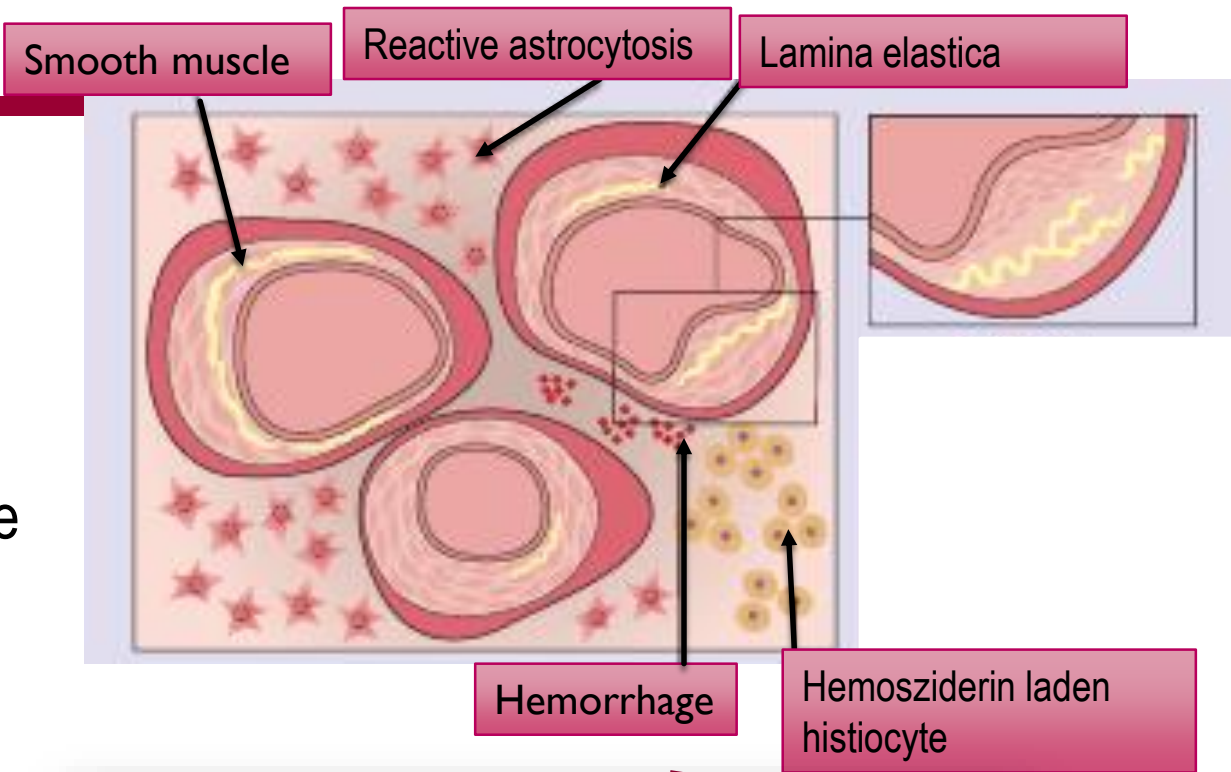
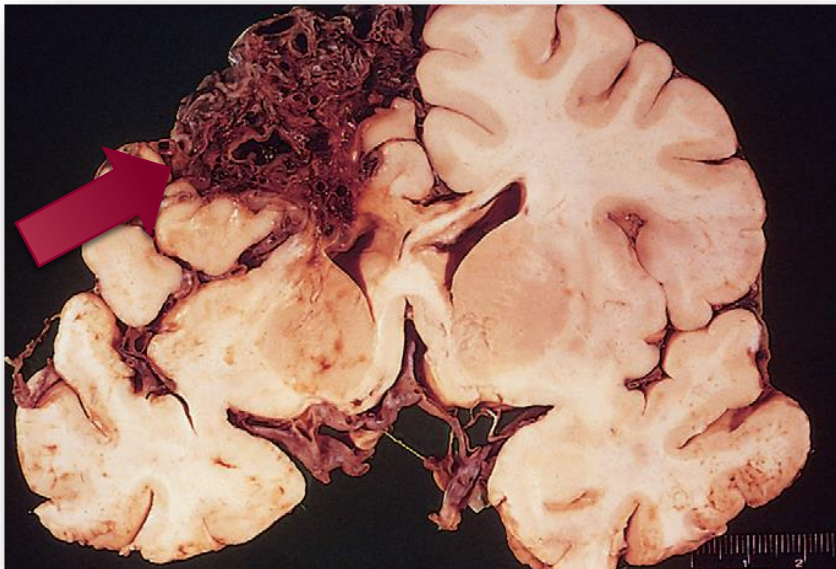
Vascular malformations

1. Arteriovenous malformation (AVM)
2. Cavernous malformation
3. Capillary telangiectasia
4. Venous angioma



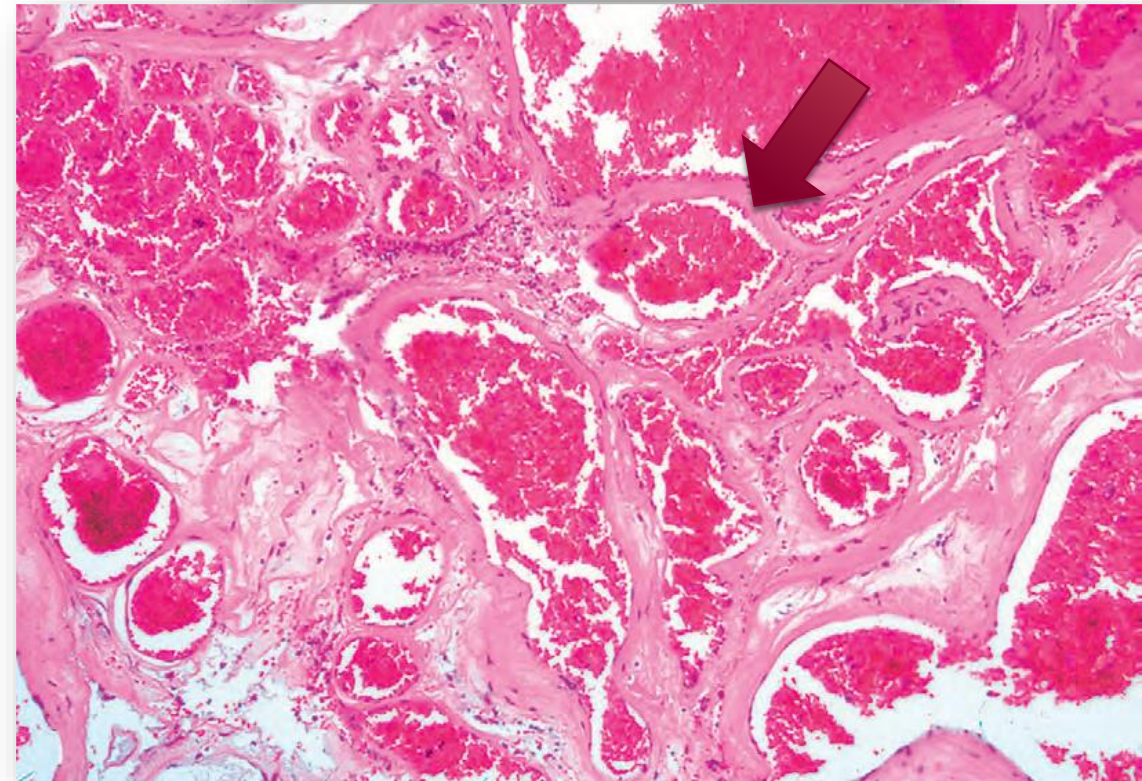
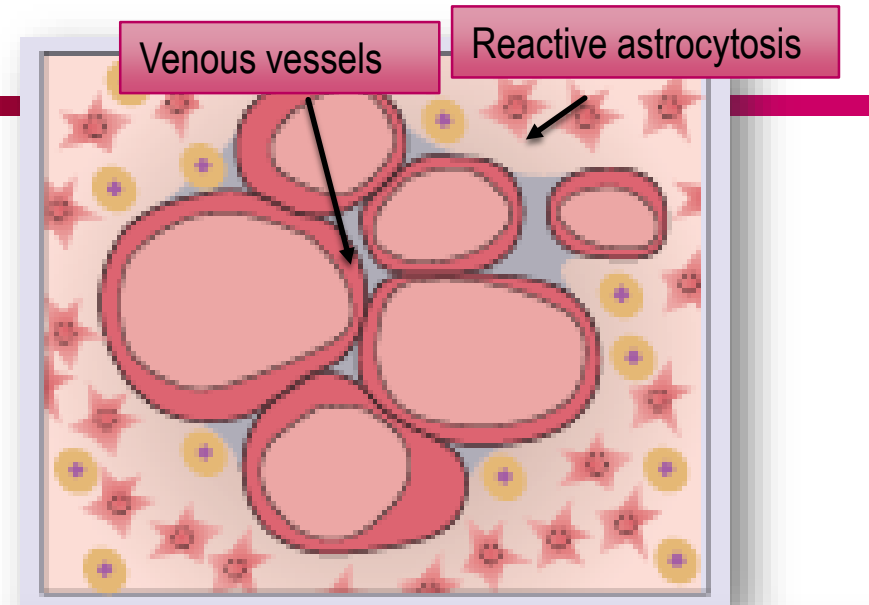
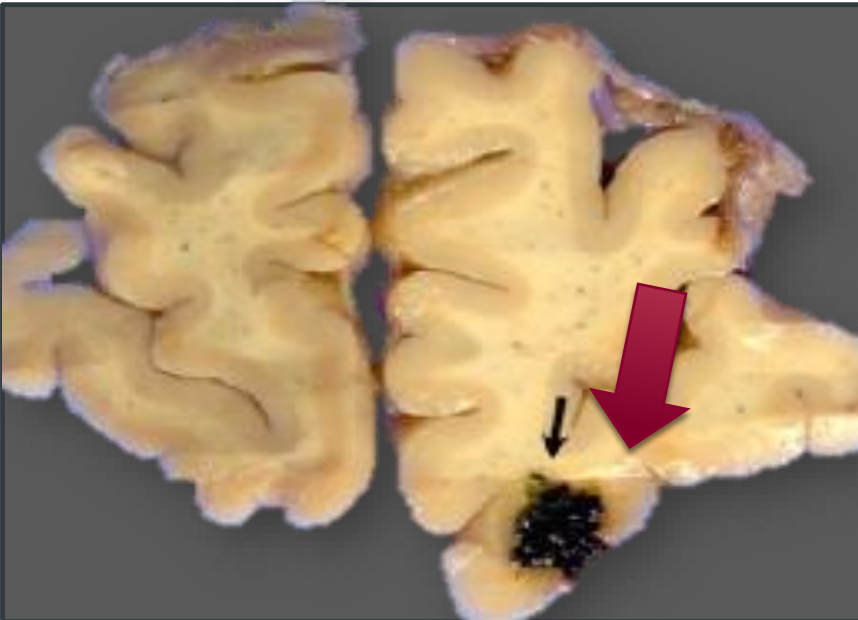
Arteriovenous malformation

- Young adults, most commonly men
- Often associated with intracerebral hemorrhage
- Arteries, veins and abnormal vessels usually embedded within brain parenchyma



Cavernous hemangioma

- Familial disorder
- Often associated with intracerebral hemorrhage
- Tightly packed collection of hyalinized vessels
- Thin venous vessels

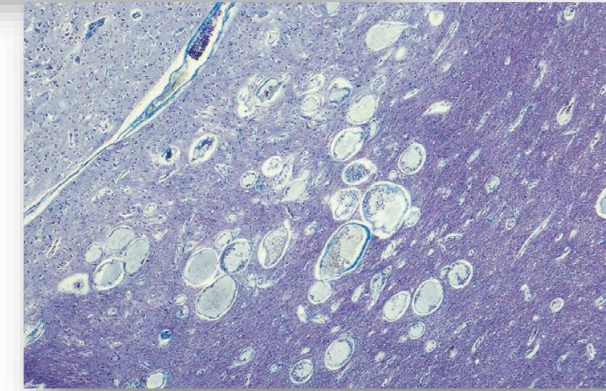
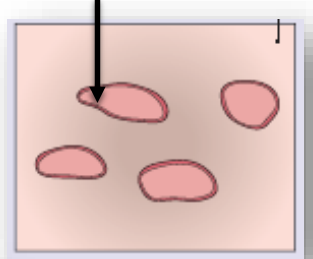


Venous angioma

- Incidental – Rarely bleed
- Cerebellum
- Thin-walled, moderately dilated vascular channels within normal brain parenchyma

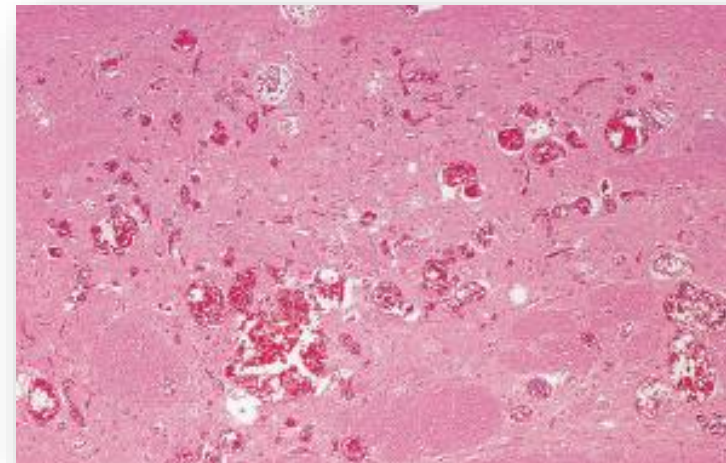


Small veins

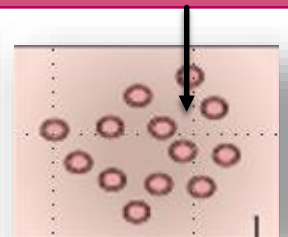


Capillary telangiectasia

- Incidental – Rarely bleed
- Small-caliber thin-walled vascular channels surrounded by brain substance

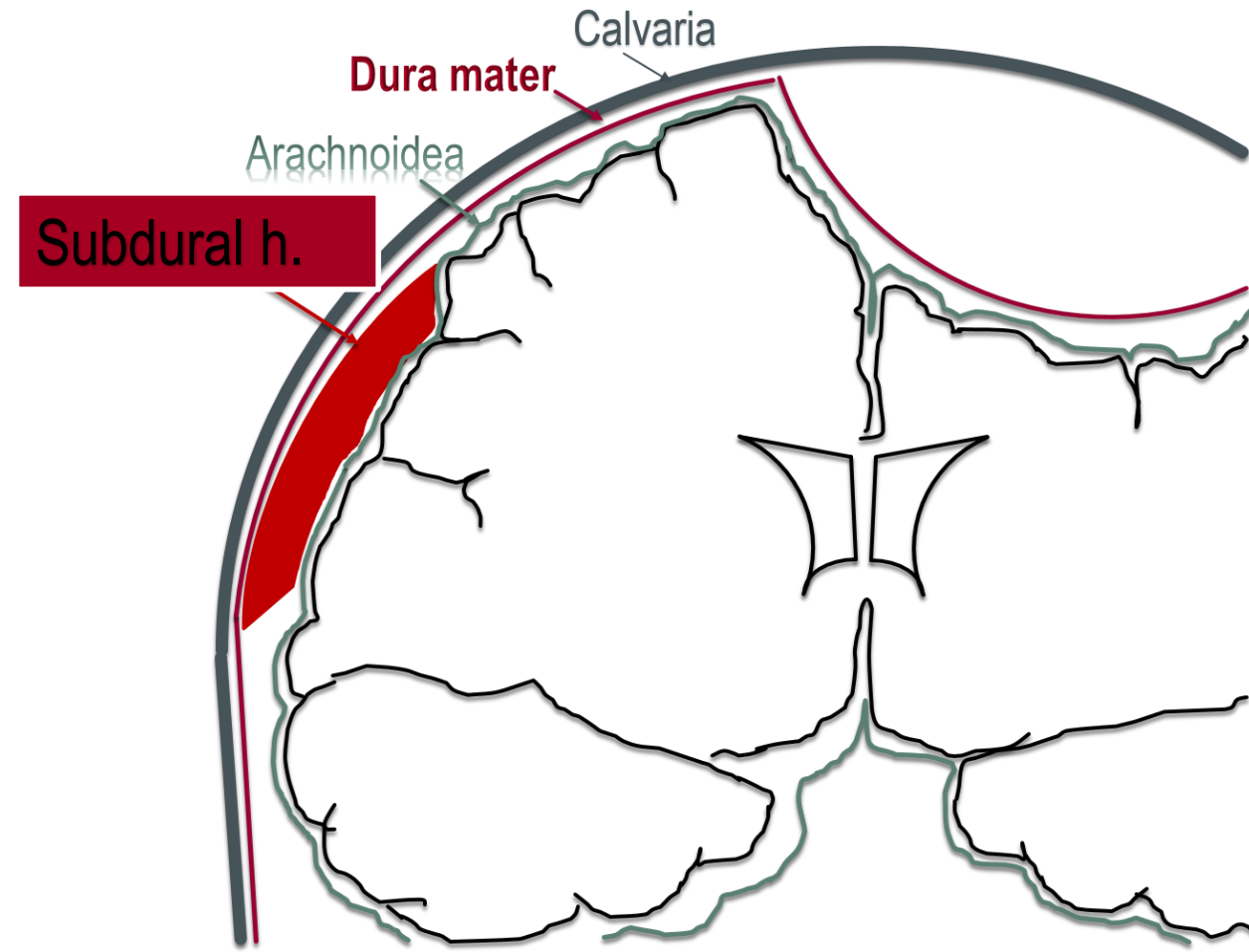
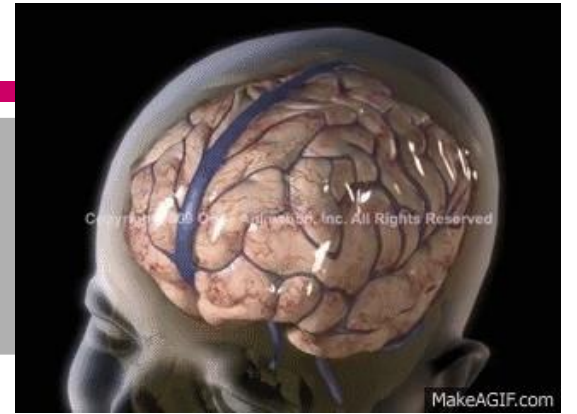
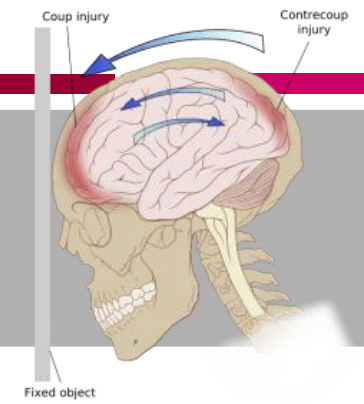
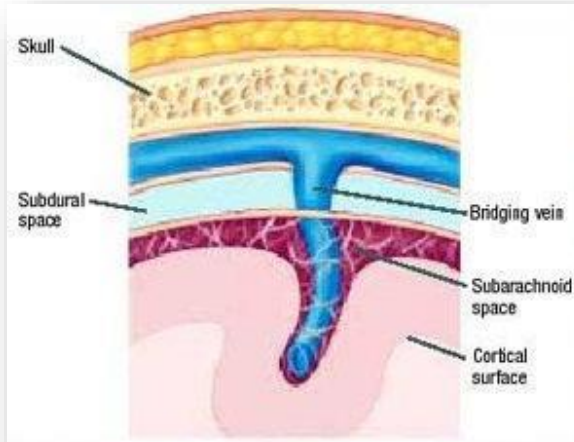


Kis kapillárisok



SUBDURAL HEMORRHAGE

- Trauma!!!
 - Rapid acceleration or deceleration
- Increased risk
 - Elderly – Brain atrophy
 - Infants – thin walled bridging veins
- Rupture of bridging veins



SUBDURAL HEMORRHAGE

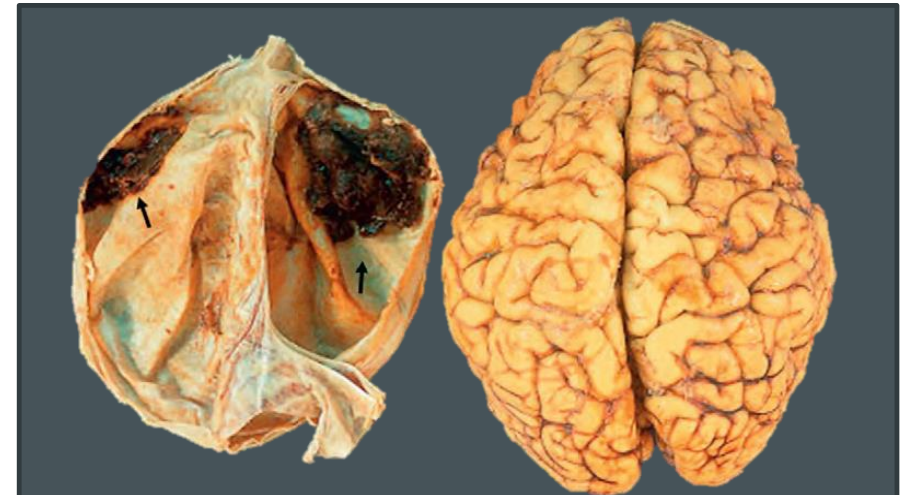
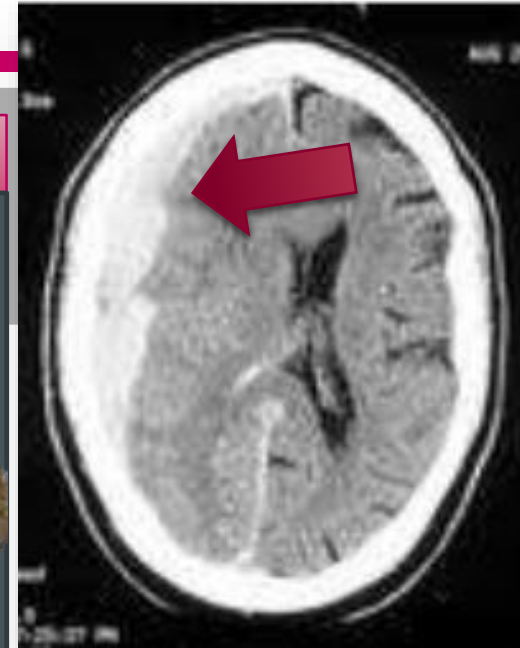
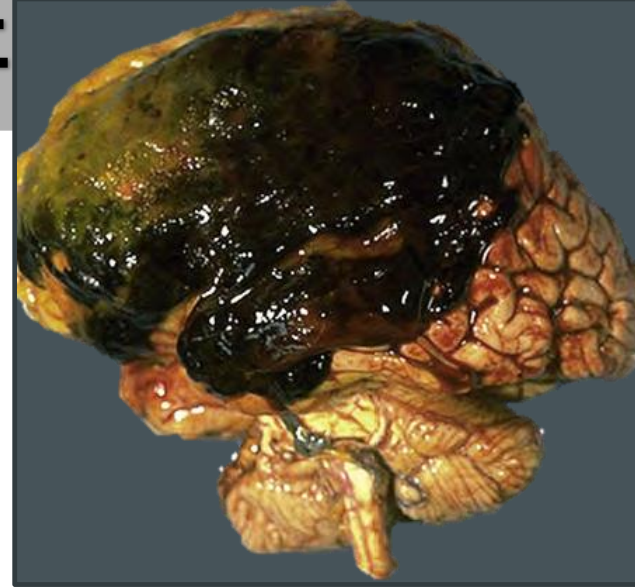
- **Acute subduralis hematoma**

- Slow progression of symptoms (manifest the first 48 hours)
- Non-localizing symptoms
 - Headache
 - Slowly progressive neurologic deterioration

- **Chronic subdural hematoma**

- Recidive trauma - Rebleeding
- Older patients
- Dementia –Alzheimer disease

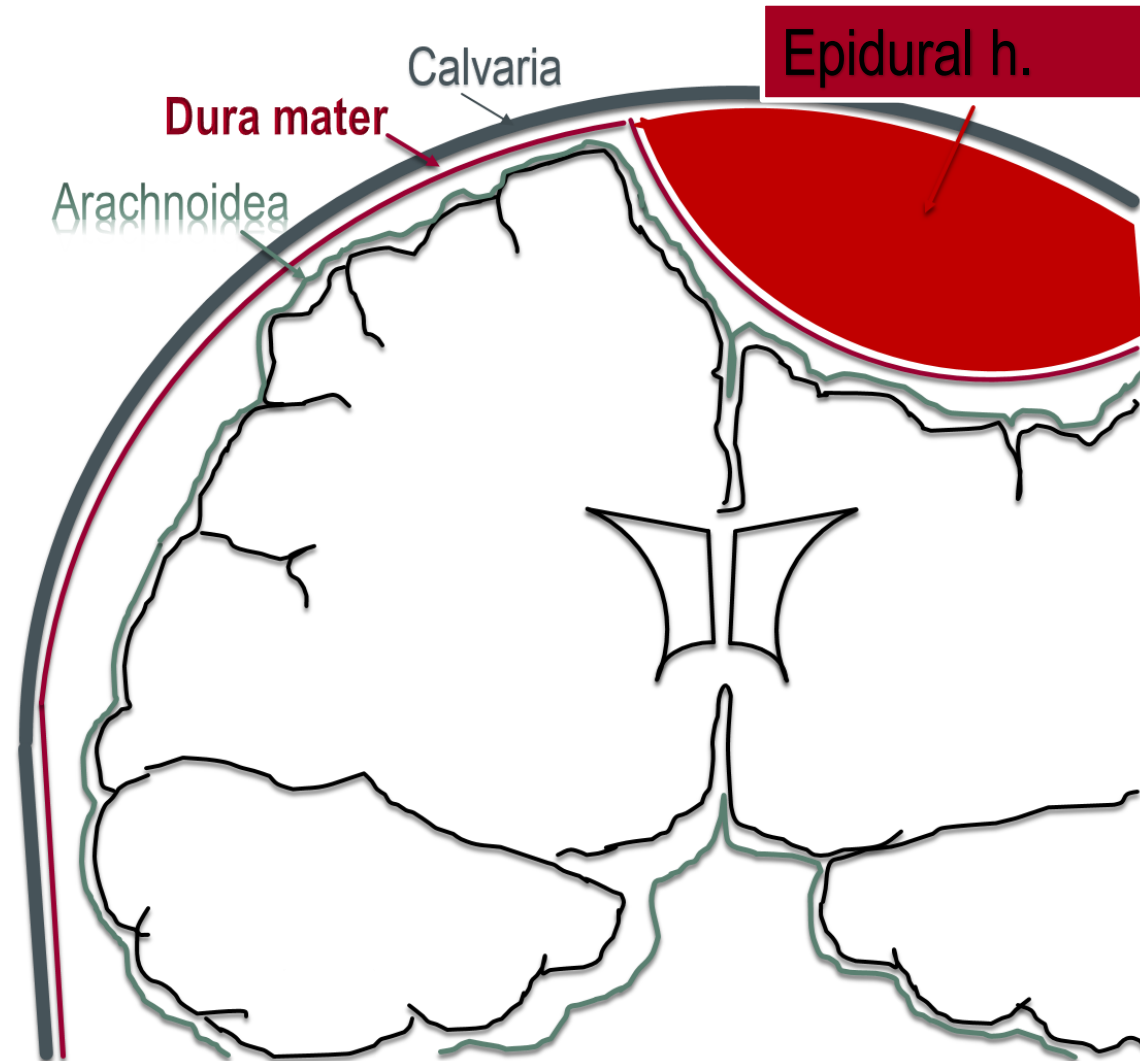
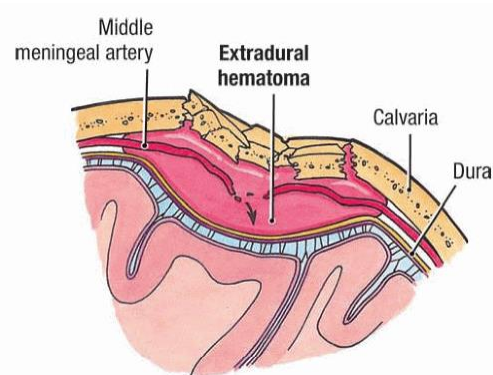
Acute subdural hematoma



Chronic subdural hematoma

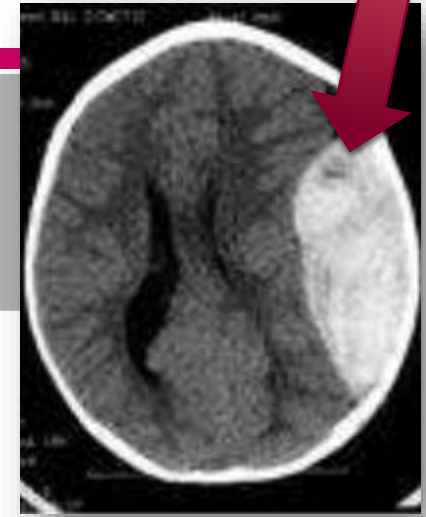
EPIDURAL HEMORRHAGE

- Trauma!!!
- Skull fracture
 - Middle meningeal artery
 - Sagittal, lateral sinus (rare)
- Adults – fossa temporalis
- Infants, children – fossa posterior



EPIDURAL HEMORRHAGE

- Arterial bleeding ~ 75-100 ml fatal
- Lucid Interval !
- Rapid progression



Skull

Acute epidural hematoma

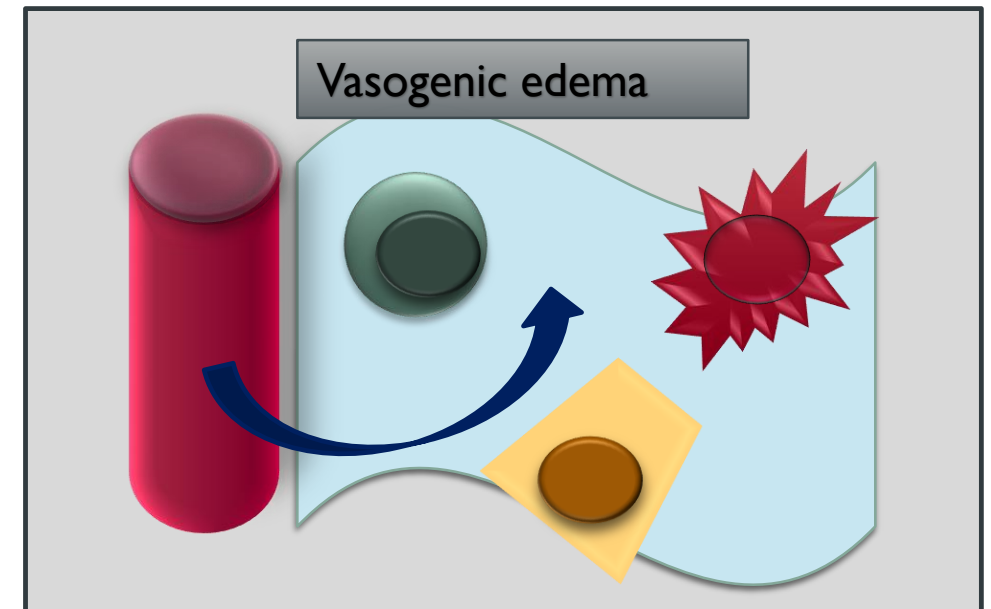
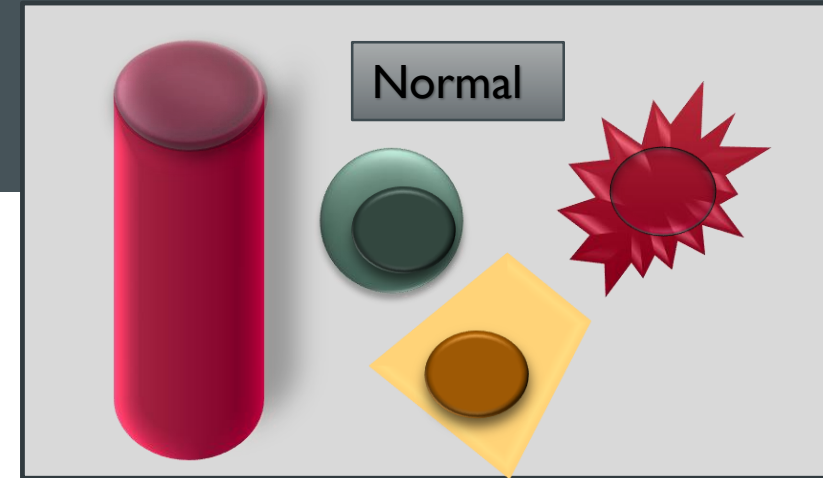
Dura mater



CEREBRAL EDEMA

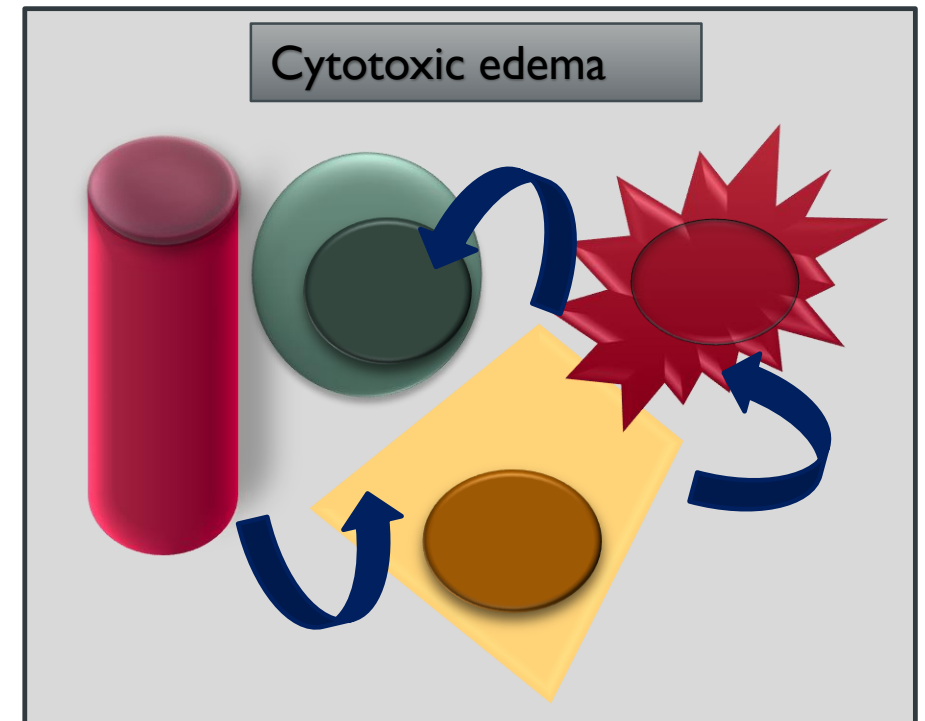
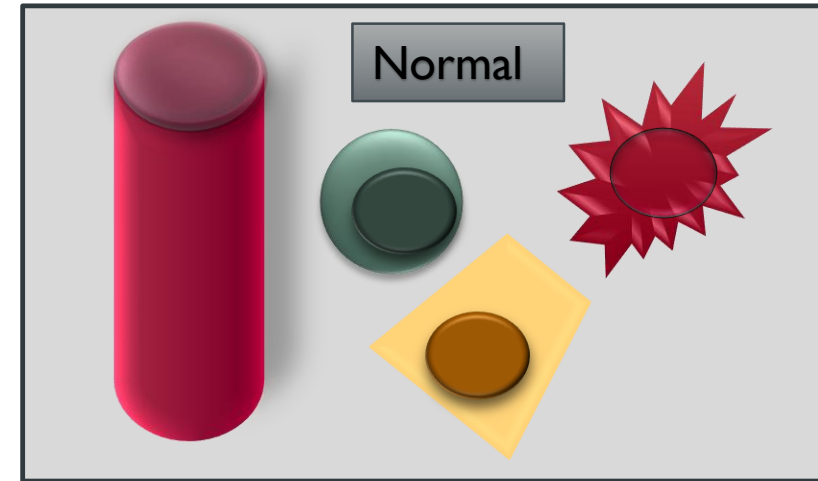
Vasogenic edema

- **Fluid shift to the extracellular compartment**
- Blood-brain barrier damage
 - Hemorrhage
 - Infarction
 - Tumor
 - Inflammation
- Affects predominantly the white matter
- Localised-generalised

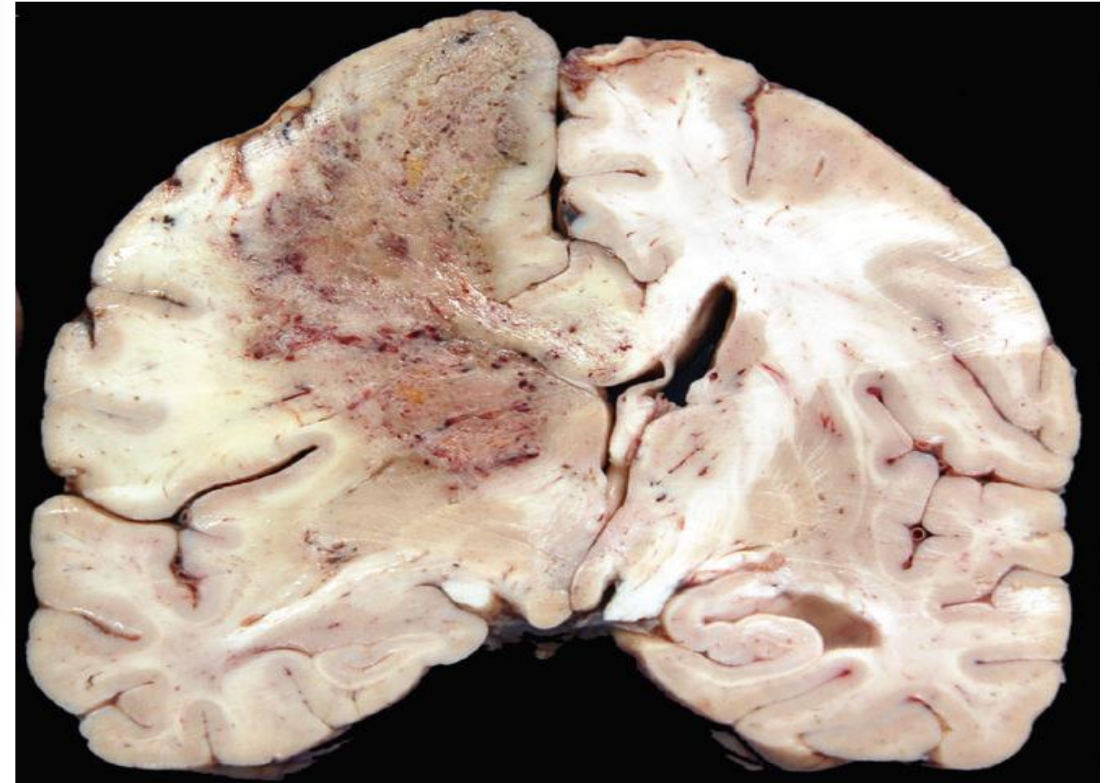


Cytotoxic edema

- **Intracellular fluid accumulation**
 - Neuron, glial cells, endothel
 - Cell membrane injury
- Cause :
 - Hypoxic-ischaemic insult
 - Toxin
 - Hypoosmolar states
 - Kidney failure
 - Diabetic ketoacidosis
- Affects the white and grey matter

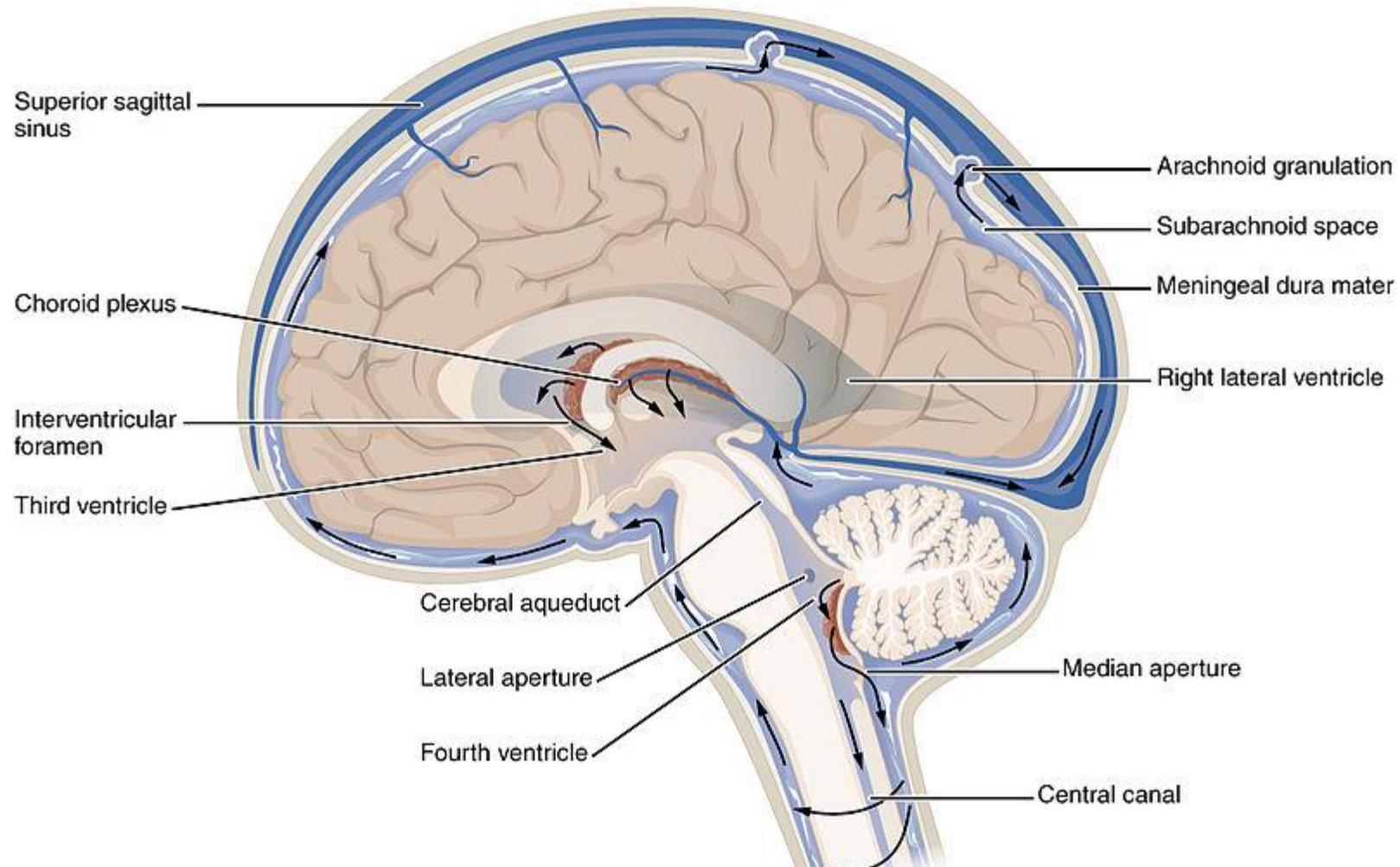


Generalised edema



Lokalised edema

HYDROCEPHALUS



Hydrocephalus:

- **The accumulation of excessive CSF within the ventricular system**

1. Communicating hydrocephalus

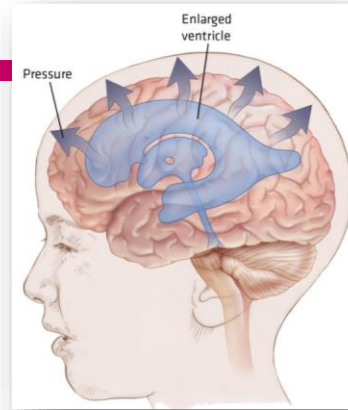
- Impaired CSF production or absorption - Rare
 - Overproduction – Choroid plexus tumor
 - Impaired resorption – Lack of arachnoideal granulations, Sinus thrombosis

2. Non-communicating hydrocephalus

- Impaired CSF circulation
 - Cause: Malformation, Tumor, Hemorrhage, Infection

3. Compensatory increase in CSF volume due to loss of brain parenchyma (Hydrocephalus ex vacuo)

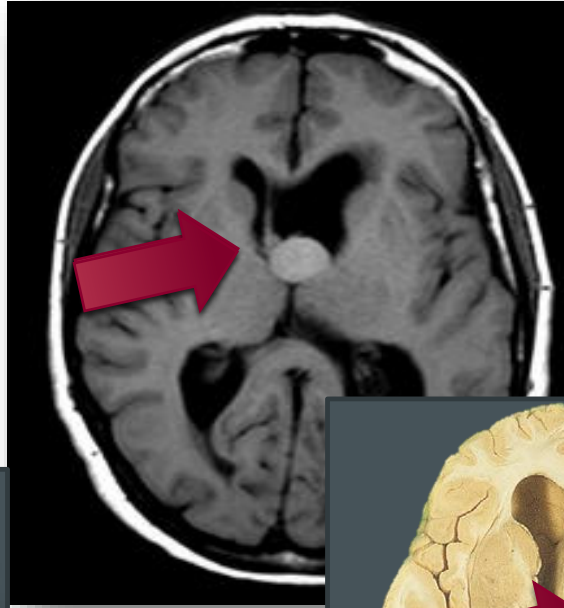
- Infarcts
- Neurodegenerative diseases



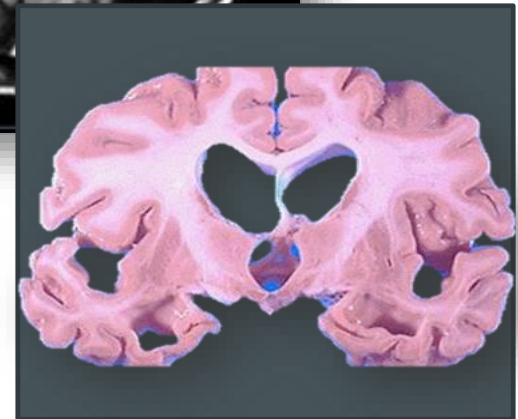
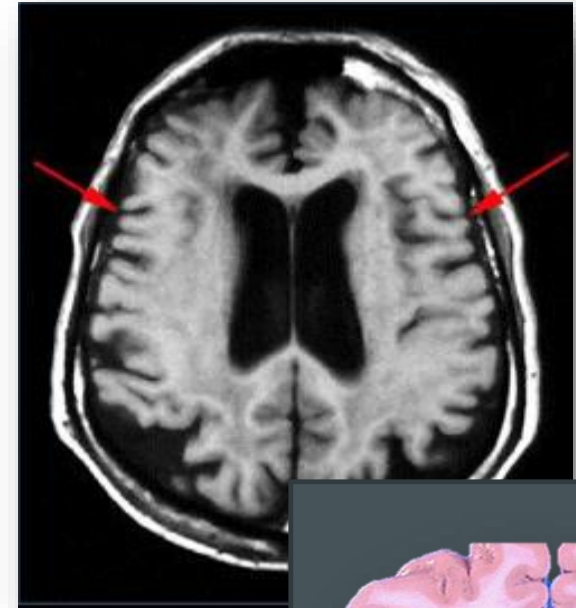
Communicating hydrocephalus



Noncommunicating hydrocephalus



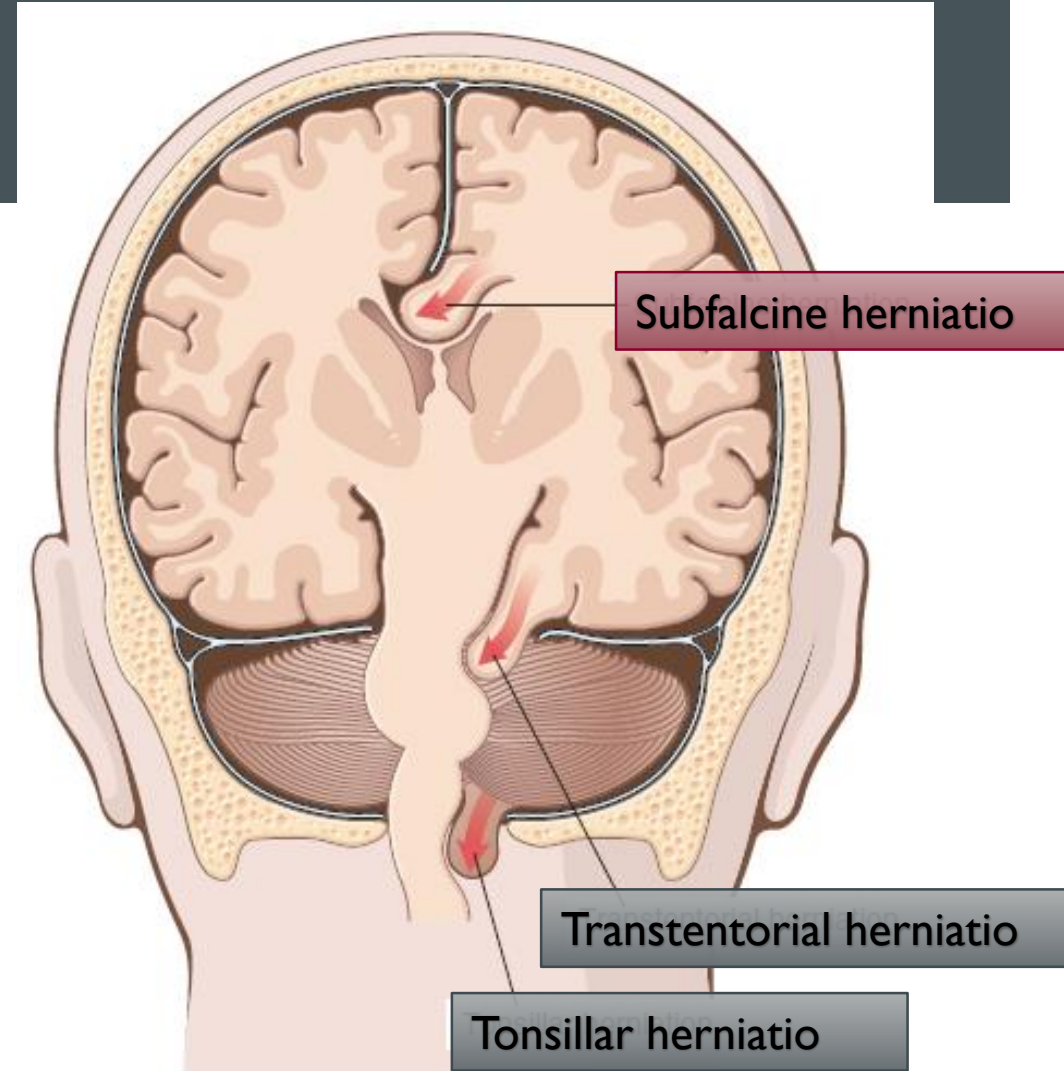
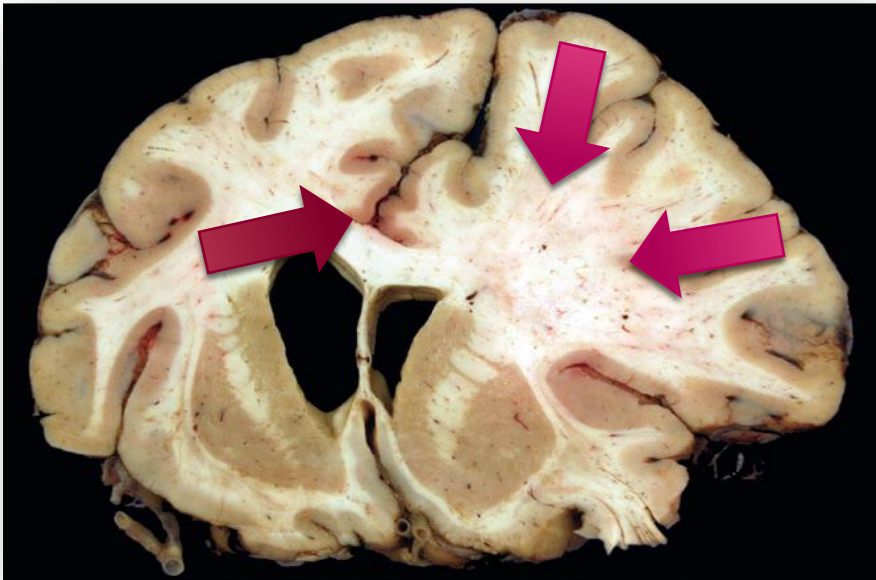
Hydrocephalus ex vacuo



HERNIATION

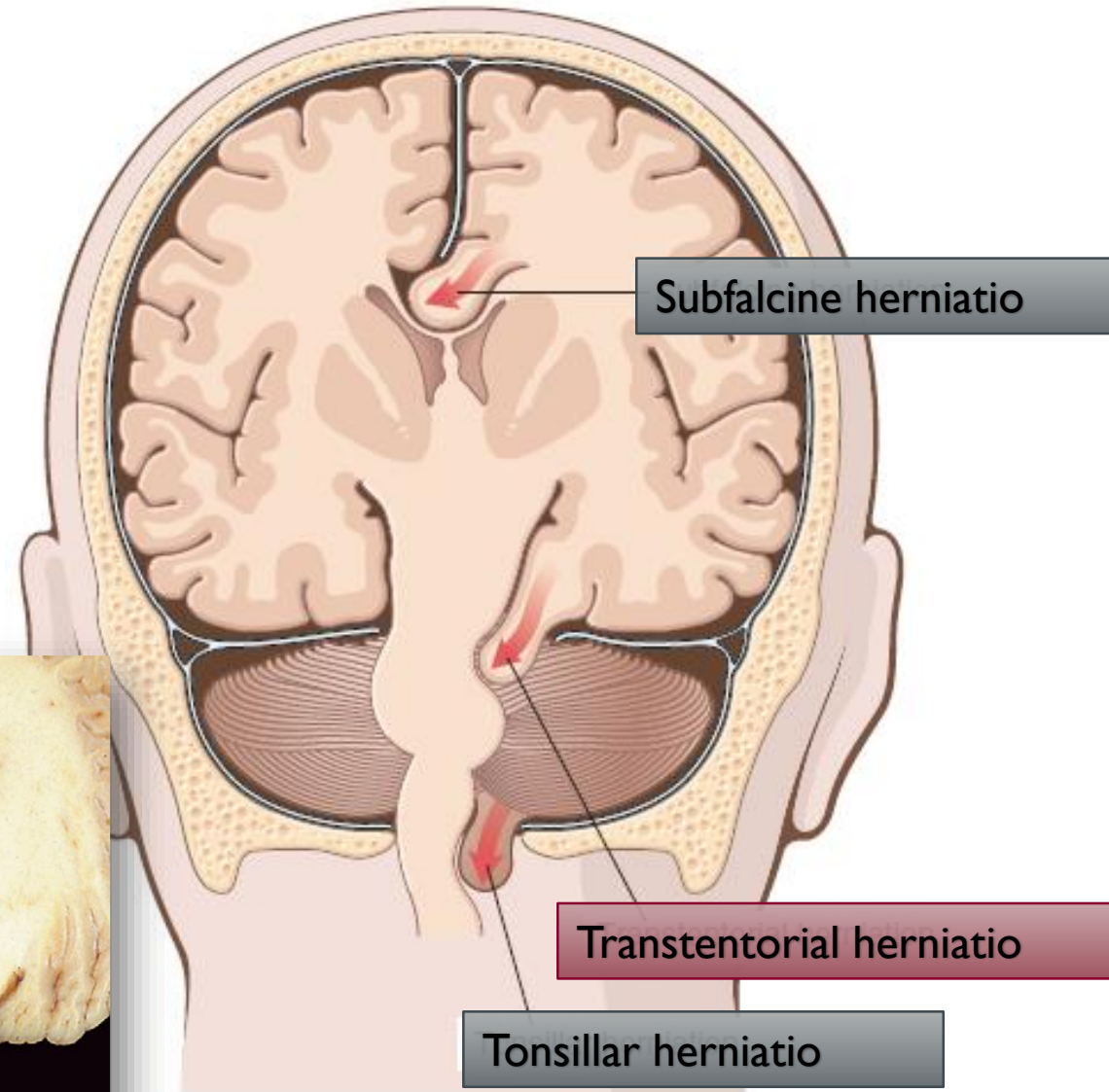
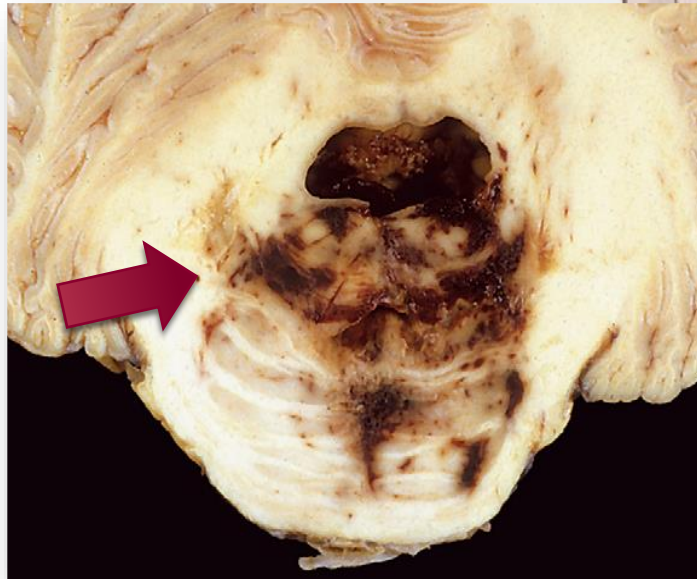
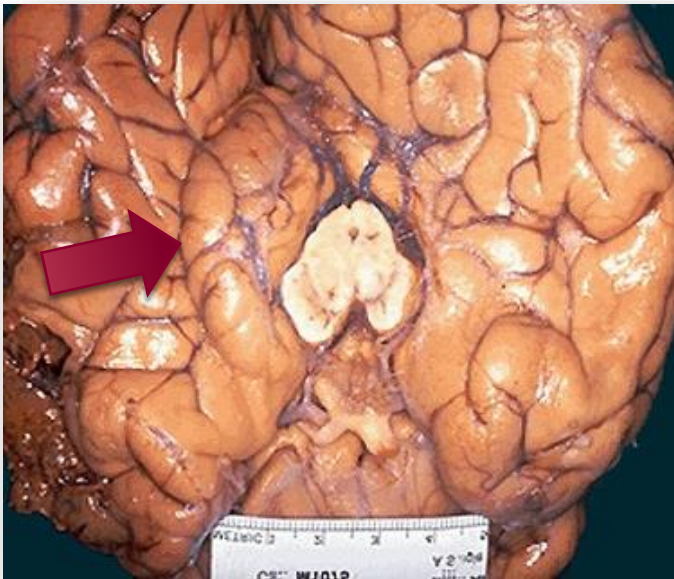
Subfalcine herniation

- Asymmetric expansion of a cerebral hemisphere
- Cingulate gyrus herniation
- Compression of anterior cerebral artery



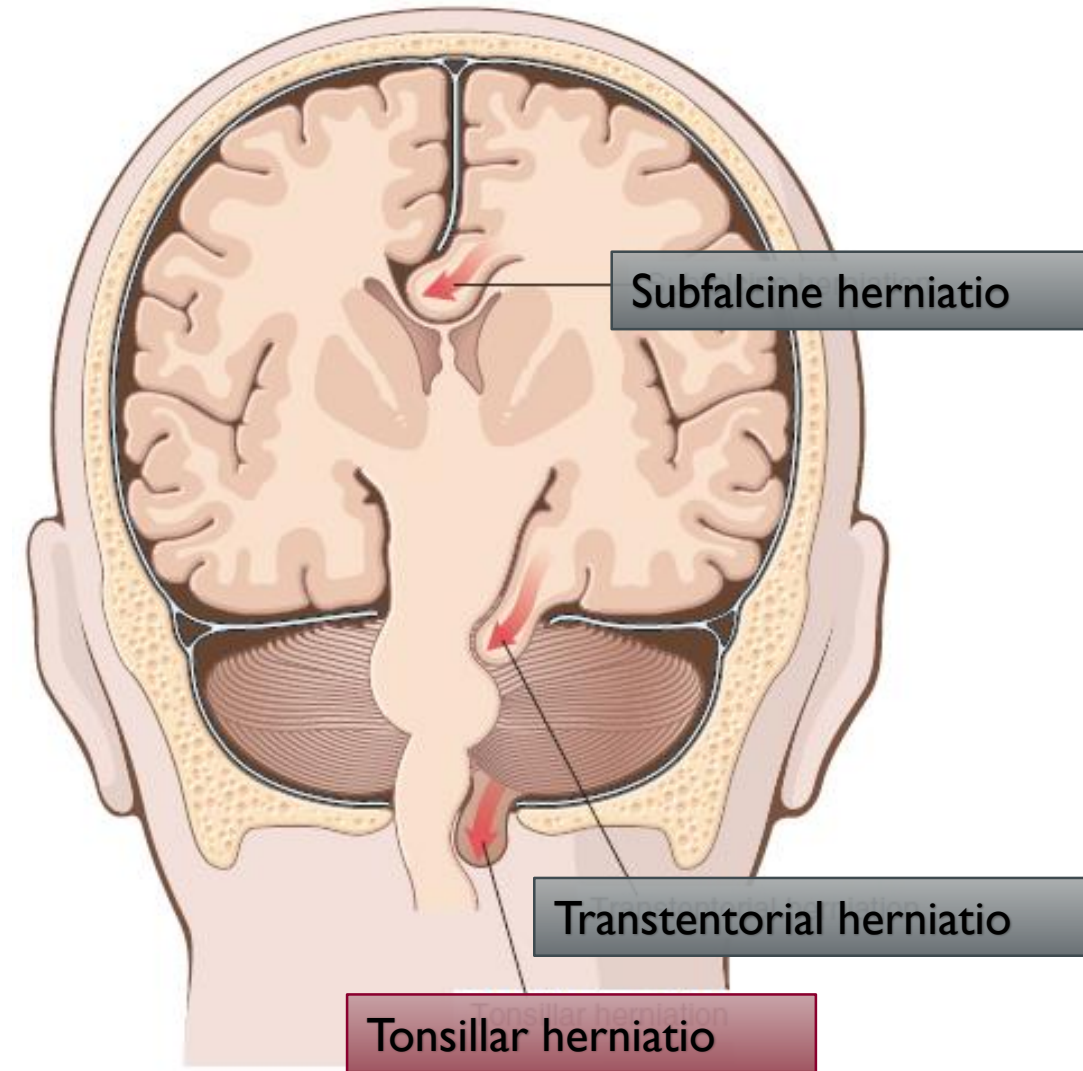
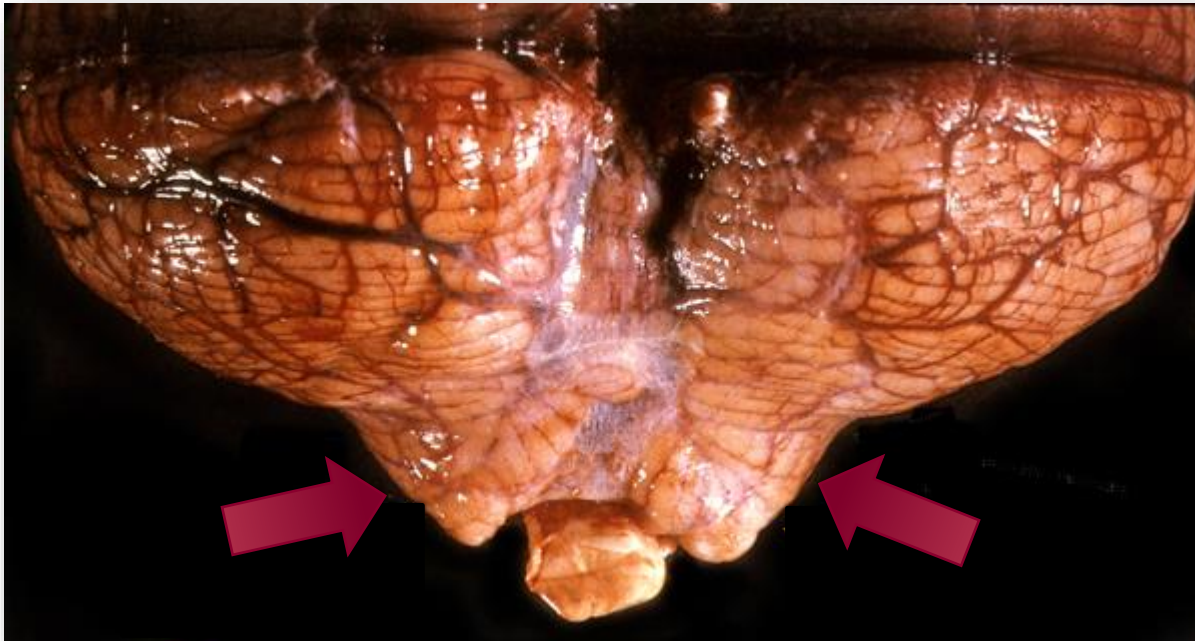
Transtentorial herniation

- Temporal lobe herniation
- Third cranial nerve and posterior cerebral artery
- Contralateral pedunculus cerebri
- Kernohan's notch
- Midbrain and pons – Duret hemorrhages

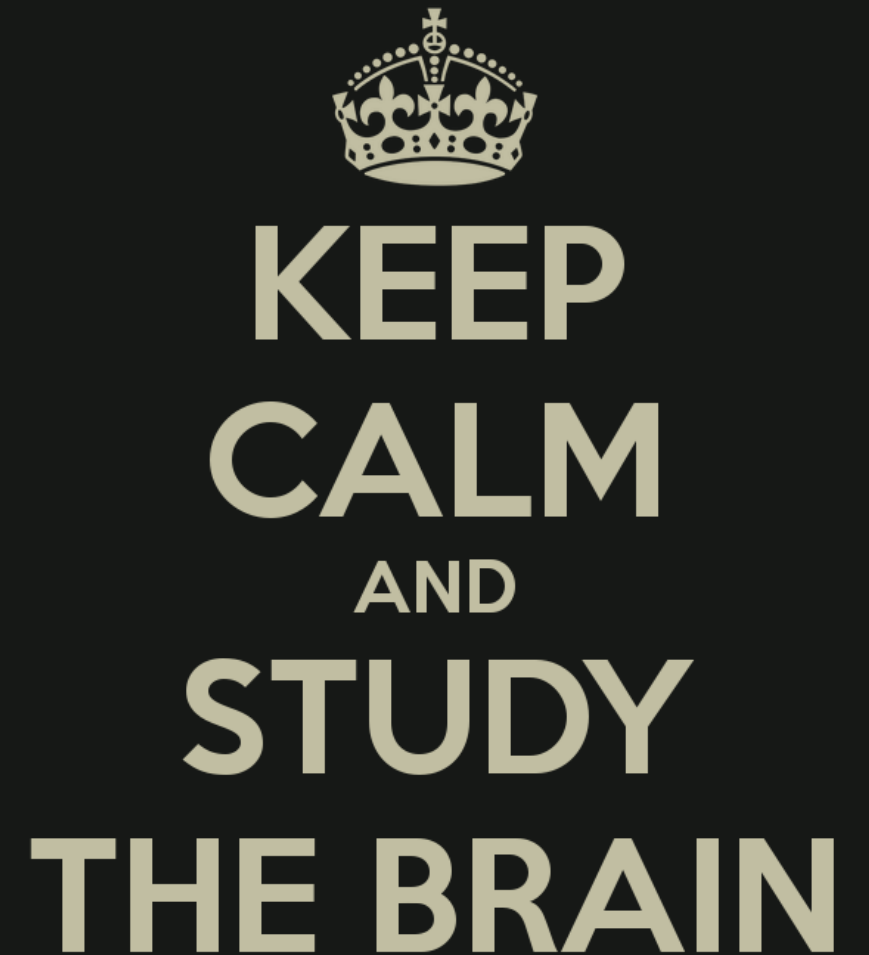


Tonsillar herniation

- Displacement of the cerebellar tonsils
- Brain stem compression
- Medulla - respiratory and cardiac centers



- Robbins Basic Pathology, 10th Edition
- Neuropathology: A Reference Text of CNS Pathology, 3rd Edition



**KEEP
CALM
AND
STUDY
THE BRAIN**